JOURNAL ARTICLE
Truth In Research (2025)

Retrofits and Revisions: How Evolutionary Theory Fails the Test of Predictive Science

Kent Hovind, Matt Nailor, Donny Budinsky.

ABSTRACT

The gold standard of science is the ability to make accurate, testable predictions. Popper (1934 -*The Logic of Scientific Discovery*) emphasized falsifiability as the defining feature of science, requiring that scientific theories make risky predictions that could in principle be proven false. While evolutionary theory is widely presented as the standard framework for biology, it has an extremely high failure rate of predictions over its conception. By contrast, the Young Earth Creationist (YEC) model, though less well known in scientific circles, offers a series of specific predictions that can be examined in light of recent data. This study evaluates the comparative predictive success of the YEC framework and evolutionary theory across multiple domains: genetic similarity, homologous genes, mutation rates, founder effects, beneficial mutations, speciation rates, functional endogenous retroviruses (ERVs), mutation saturation, and the unity of the human race. We review published data and analyze whether these observations more closely align with YEC predictions or evolutionary expectations. Our analysis suggests that most observed outcomes are more consistent with the YEC predictive model than with evolutionary predictions, particularly regarding homologous genes, genetic similarities, hierarchical patterns, mutation rates, limits to beneficial mutation, endosymbiosis, multicellularity and the functional role of so-called “junk” DNA such as endogenous retroviruses (ERV’s). We conclude by discussing the implications of these findings for the broader question of falsifiability in scientific theory, and argue that the YEC framework warrants further consideration as a predictive scientific model.

INTRODUCTION

Predictions on Trial: Evolution vs. Creation. Genetic similarity across diverse organisms is often presented as a key prediction of evolutionary theory. Yet historically, leading biologists such as Ernst Mayr anticipated that conserved genes would be virtually undetectable across distant taxa, given the presumed accumulation of random mutations over deep time. Contrary to this expectation, discoveries such as Hox clusters and conserved regulatory networks revealed striking similarities across insects, mammals, and other groups. These findings, initially unexpected, were later incorporated into evolutionary explanations, even though creationist researchers had earlier predicted such patterns based on common design.

The history of this debate highlights a broader pattern: when new, surprising genetic or anatomical findings emerge, which contradict previous predictions made by evolution theory, they are often retroactively fit into evolutionary models rather than prompting reconsideration of alternative hypotheses. By contrast, such findings are consistent with design-based frameworks and predictions that flow from the Biblical Model of ancestry based on scripture.

Our goal is not merely to rehearse old arguments or existing debates, but to bring together multiple lines of empirical evidence—molecular, developmental, genetic and population-level—biology that repeatedly exposes the shortcomings of evolutionary predictions while aligning with testable, design-based expectations that have been confirmed across disciplines, offering a more powerful and consistent explanation for the diversity of life on earth."

In the following sections, we evaluate some of the most frequently cited examples of supposed evolutionary novelty—such as taxonomy, phylogeny, the tree of life, beneficial mutations, genetic similarities, nested hierarchies, mutation saturation and show that these so called best pieces of evidence for evolution are not actually evidence for evolution at all but rather creationist predictions. Then we delve into the Hox gene mutations to expose the reality of the results. Even the Italian wall lizard, often paraded as proof of new structures, instead showcases regulatory plasticity and latent design—and assess whether these cases demonstrate genuinely new anatomical structures or instead reflect regulatory flexibility and latent genetic capacity. We further examine pedigree-based mutation rate studies and the emerging roles of endogenous retroviruses and epigenetic mechanisms, showing that these findings consistently undermine long-age evolutionary models while aligning with testable predictions of recent creation.

EVIDENCE

We begin our journal with Carl Linnaeus, the Swedish botanist and physician known as the "Father of Taxonomy." Like Owen, Linnaeus worked from an Anglican creationist framework. He saw his work of classifying the diversity of plants and animals not as uncovering evolutionary relationships, but as "*thinking God's thoughts after Him*" by uncovering the order God placed in creation.

Linnaeus's binomial nomenclature system (*Systema Naturae*, 1735–1758) was rooted in the belief that species were created separately, each according to their kind (*Genesis 1*). His writings reveal that he initially believed in the immutability of species but later recognized the possibility of limited variation within created kinds — a key distinction also affirmed in modern creation biology.

> He wrote: "*The Earth's creation is the glory of God, as seen from the works of Nature by Man alone. The study of Nature would reveal the Divine order of God's creation.*" (*Systema Naturae*, Preface, 1735).

Linnaeus' Predictions

1. ***Order and Hierarchy in Creation***

- **Prediction** (1735–1758): Linnaeus predicted that organisms could be classified into fixed, natural groups (kingdom, class, order, genus, species) because God created them with a rational order.
- He saw taxonomy not as man-made convenience, but as a reflection of real categories placed into nature by God Himself.

Quote: "*God created, Linnaeus arranged.*" *(Inscription on Linnaeus's statue in Uppsala, summarizing his view).*

Confirmation:

Comparative Anatomy

Confirmed by the pioneering work of Sir Richard Owen, comparative anatomy established that organisms share a common structural plan, yet within clear anatomical limits. In many ways, Owen's discoveries confirmed Linnaeus's earlier predictions that nature was organized into real, discoverable categories. Far from illustrating gradual evolutionary transitions, comparative anatomy reveals *boundaries of form* — structural discontinuities that point to a creation-based pattern of organization.

Taxonomy and the Boundary Paradox

Taxonomy remains the indispensable framework of modern biology. Despite layers of evolutionary reinterpretation, the Linnaean categories *(kingdom, phylum, class, order, family, genus, species)* remain foundational and irreplaceable. This is because they reflect real, observable groupings in nature. Linnaeus's original system emphasized bounded groups (created kinds), whereas evolutionary theory requires an open continuum with no absolute barriers. This tension persists as the "taxonomic boundary paradox": classification works only because of the very boundaries that evolutionary theory struggles to explain.

Baraminology

Building on Linnaeus's recognition of created "kinds," **baraminology** (from Hebrew *bara* = "created" and *min* = "kind") investigates the limits of biological variation. Empirical studies across plants, animals, and microbes consistently demonstrate a twofold pattern:

1. **Variation within kinds** — populations display real adaptability and diversity seen through phenotypic adaptations most through epigenetic factors.

2. **Boundaries between kinds** — lineages remain fixed within deep divisions, showing no evidence of blending across kinds.

This confirms the creation model of distinct baramins rather than a seamless chain of common descent.

Genomics (21st Century)

With the advent of whole-genome sequencing, boundaries once evident only in taxonomy and anatomy have now been confirmed at the genetic level. A landmark 2018 study (Thaler et al) demonstrated that genomic data clusters organisms into discrete groups, not a smooth evolutionary continuum. These findings confirm not only Linnaean and baraminological predictions but also Owen's anatomical observations: boundaries exist across morphology, anatomy, and now genetics.

Together, these disciplines — comparative anatomy, taxonomy, baraminology, and genomics — converge to confirm the **creation-based expectation of fixed boundaries**. Far from supporting evolutionary gradualism, the evidence consistently reinforces the presence of discrete, divinely ordered categories.

2. Species Created According to Their Kind

   Prediction: Linnaeus predicted that God created "kinds", and that there were boundaries between "kinds" and variations within those kinds. This is why he created taxonomic boundaries and classifications.

Quote: "Species are the works of nature, varieties are the works of time." (*Systema Naturae*, 10th edition, 1758).

Confirmation:

- Genetics (20th century): Modern biology confirms microevolutionary changes within kinds (allele shifts, hybridization) but no evidence of macroevolutionary transformations across boundaries. Evolution has now had to alter the definition and embrace that micro-changes must lead to macro-changes over deep time, abandoning earlier predictions. The linear evolutionary tree is now discussed only in terms of the tips of the branches through **descent** with modification, exactly as he predicted we would find.

- Creation biology interprets this as **variation within created kinds**, exactly as Linnaeus predicted and the taxonomic boundary paradox still plagues evolution theory to this day since there are no boundaries in evolution.

3. Man as a Unique Creation

   Prediction: Linnaeus classified humans among primates but with zero intent on relation, rather he insisted on man's unique linguistic, spiritual and moral status. He predicted that biology alone would never erase the distinction between humans and animals.

Quote: *"Man stands alone."* (Systema Naturae, 1758, under Homo sapiens).

Confirmation:

- Anthropology and neurology continue to confirm sharp boundaries between humans and animals in language, morality, and abstract thought.

- Even in genetics, the degree of similarity is greater than predicted by evolutionary assumptions of common ancestry and deep time.

Our journey continues with one of the most frequently cited evidences for evolution: similarity across diverse organisms. It is often assumed that this similarity was an evolutionary prediction based on relation; however, historical sources prove otherwise. Sir Richard Owen, the 19th-century anatomist and creationist, proposed the idea of the "archetype" in comparative anatomy. His archetype was not an evolutionary concept—it was a conceptual blueprint showing a common structural plan underlying vertebrates, particularly the skeleton based on common design. Owen made several predictions based on this archetype, such as the presence of certain bones in the limbs of vertebrates, even if they appeared modified or missing in some species. He stated: *"The archetype points to a deep and pregnant principle...some archetypal exemplar on which it has pleased the Creator to frame certain of his living creatures."*

Owens Predictions

1. Homologous bones in vertebrate limbs

Prediction (1830s–1840s): Owen predicted that all vertebrates would share the blueprints for a common pattern in their limb bones: one upper bone (humerus/femur), two lower bones (radius & ulna / tibia & fibula), wrist/ankle bones, and digits—even if some are reduced or modified. His exact phrasing often used "unity of type": "*The same essential parts of the skeleton are found under every variety of form and modification, throughout the whole series of vertebrated animals.*" (On the Nature of Limbs, 1849).

Confirmation:

- Embryology (late 19th–early 20th century): Limb buds in vertebrate embryos showed the same basic structure.

- Genetics (1978): Discovery of Hox genes controlling limb patterning confirmed a shared developmental blueprint.

2. Vertebral structure

Prediction: Owen suggested that vertebrae, ribs, and related skeletal elements follow a common structural archetype in all vertebrates. His exact phrasing was: "*The general proposition, therefore, is, that the bony and cartilaginous parts of the skeleton of the Vertebrata are homologous throughout the group; and that the modifications of these parts in the different animals admit of being traced to deviations from a common type.*" *Owen suggested that:*

- *The* ***vertebra*** *was the fundamental structural unit of the skeleton.*

- *Ribs, limb girdles, and even parts of the skull could be interpreted as **modified vertebrae or vertebral appendages.***

- *Thus, all vertebrate skeletons could be mapped back to a **single archetypal blueprint.***

"Every segment of the endo-skeleton consists of a series of parts homologous with those of a vertebra." (On the Archetype..., 1848)

Confirmation:

- Comparative anatomy (19th century): Anatomists found homologous vertebrae across fish, reptiles, mammals, and birds.

- Embryology & Genetics (20th century): Cytochrome C gene studies in conserved regions confirmed that vertebral identities are genetically preserved.

His accurate prediction that all vertebrate skeletons follow this "vertebra-based plan" provided the intellectual framework later plagiarized by evolutionary theory to argue for common ancestry.

3. Skull elements & Limb element reduction and modification

Prediction: Owen's archetype theory proposed that the vertebrate skull is built from repeated segments, corresponding to vertebrae and arches. Even "missing" or highly modified bones (e.g., whale flippers, bird wings) are part of the archetype—they're just modified.

Confirmation:

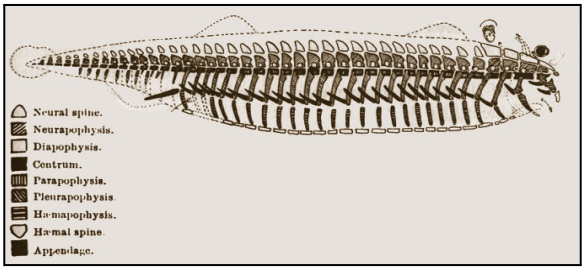
- Late 19th–early 20th century: Anatomists confirmed skull homologies between fish, amphibians, reptiles, birds, and mammals. Comparative embryology confirmed rudimentary elements in embryos.

- Molecular/developmental biology (1970s–1980s): Neural crest and HOX gene studies validated a conserved developmental plan for the skull. Limb development showed the same genes specify these elements, even if they don't develop fully in adults.

Before his predictions were all confirmed, Darwin poked fun at this in Origin of Species (1859), saying Owen's archetype was like a *"mystical ideal"* that explained nothing. Ironically Darwin later stole Owen's ideas and twisted them into his own version to use homology as evidence for evolution. A tricky move for anyone not paying attention. He even took Owen's concept of "Similarity due to common design", and changed it to "Similarity due to common ancestry." A common tactic by Darwin as you will see later.

Then in another evolutionary surprise, Owen's description of an Archetype was actually discovered in 2012. *"For decades, scientists believed that a spine with multiple segments was an exclusive feature of land-dwelling animals."* (*Phys.org*, May 23, 2012)

Image 1. *Owen's Archetype* **Image 2.** *Tarrasius problematicus*



Sir Richard Owen, working from a creation framework, predicted that life would display a nested hierarchical pattern because God had created according to an ideal structural plan. He described this as *"that ideal original or fundamental pattern on which a natural group of animals or system of organs has been constructed, and to modifications of which the various of such animals or organs may be referred"* (Owen, 1848). Rather than evolution producing branching continuity, Owen saw the nested pattern as the outworking of a biblical principle of design. Scripture records that God made creatures "after their kind" (Genesis 1), each reflecting variations from a single archetypal design. For Owen, the nested hierarchy in nature was not evidence of common descent but confirmation of a common Creator (Image 3).

Image 3

So while Owen made his predictions in the 1830s–1840s, the real confirmation did not arrive until the 1960s–1970s, when molecular and developmental biology revealed a **conserved genetic program** underlying vertebrate morphology. And here the irony deepens: what should have strengthened evolutionary theory instead exposed its weakness, showing that the very framework long claimed as evidence for descent was better explained as evidence of **common design**.

DNA Similarity: Evolution's Failed Prediction, Creation's Fulfilled Expectation

In 1963, Harvard evolutionary biologist Ernst Mayr argued that searching for conserved DNA similarities between very different organisms would be unproductive, since random genetic changes accumulated over millions of years would likely obliterate and obscure such similarities.

He stated *"Much that has been learned about gene physiology makes it evident that the search for homologous genes [similar codes due to common ancestry] is quite futile except in very close relatives. If there is only one efficient solution for a certain functional demand, very different gene complexes will come up with the same solution, no matter how different the pathway by which it is achieved. The saying "Many roads lead to Rome" is as true in evolution as in daily affairs."* [1] (Mayr, 1963, as cited in Gilbert et al., 2000, p. 609).

On the creationist side, predictions regarding genetic similarity also predated detailed DNA analyses, and went off prior creationist predictions on similarities in both homology (Sir Richard Owen's 1843) and taxonomy (Carl Linnaeus 1745). In 1975, Henry Morris (*founder of the Institute for Creation Research*) argued that both homologous genes would be found across most forms of life, and genetic similarity in DNA would be reflecting common functional requirements and design patterns in his book *The troubled waters of evolution* (1975).

He said: *"The creative process would have designed similar structures for similar functions and different structures for different functions... In the creation model, the same similarities are predicted on the basis of a common purposive designer."... "It may yet be demonstrated that all living organisms exhibit similar underlying DNA-level properties reflecting a unified pattern of functional design. Such findings would be consistent with the hypothesis of a common designer and suggest that relatively little time has elapsed since creation"* (Morris, 1975).

Regarding the first part of his prediction, in 1978, Edward Lewis confirmed the first YEC prediction by Morris and demonstrated that Hox genes in *Drosophila* are arranged in clusters along the chromosome and that their order corresponds to their expression pattern along the anterior–posterior body axis (Maeda et al., 2009, reviewing Lewis's work). This discovery established that a conserved genetic regulatory system underlies body-plan development. Later research has shown that Hox clusters and their functions are not unique to fruit flies but are widespread across the animal kingdom, including vertebrates such as mice, humans, and elephants. Developmental biologist Sean Carroll has highlighted the significance of this finding, noting that the universal distribution of Hox genes was unexpected even among proponents of evolutionary theory (Carroll, 2005). Darwin's prediction *(plagiarization*) was not the pattern (the hierarchy), but the mechanism (natural selection, descent with modification).

Subsequent developmental biology research has demonstrated that conserved genetic regulatory information is widespread across diverse animal groups and underlies the development of similar anatomical structures. In his synthesis of evo-devo research, Sean Carroll emphasized the significance of the discovery: *"It was inescapable. Clusters of Hox genes shaped the development of animals as different as flies and mice, and now we know that includes just about every animal in the kingdom, including humans and elephants. Not even the most ardent advocate of fruit fly research predicted the universal distribution and importance of Hox genes"* (Carroll, 2005, p. 64). Carroll further observed: *"The implications were stunning. Disparate animals were built using not just the same kinds of tools, but indeed, the very same genes... No biologist had even the foggiest notion that such similarities could exist between genes of such different animals"* (Carroll, 2005, pp. 64–65). In a later reflection, he noted that this represented a failure of evolutionary expectations: *"Comparative and evolutionary biologists had long assumed that different groups of animals, separated by vast amounts of evolutionary time, were constructed and had evolved by entirely different means"* (Carroll, 2018). These acknowledgments illustrate the **unexpected** nature of Hox gene conservation across animal phyla and highlight the challenge such findings posed to prior evolutionary assumptions.

Frederick Sanger's development of DNA sequencing in 1977 two years after Morris' prediction finally made it possible to directly read the genetic code, allowing scientists to compare genes — not just proteins (Dayhoff 1960s) across species. This breakthrough opened the door to modern genomics and provided the tools that confirmed Morris's prediction of underlying DNA-level similarities across life. By contrast, evolutionary theorists like Motoo Kimura, writing around the same time, assumed any such similarity as the product of random genetic drift rather than purposeful design. In effect, Kimura predicted the exact opposite of Morris: *that shared DNA sequences reflected chance processes, not functional patterns.* Today, the evidence is clear: genetic sequences are not random strings but carry functional purposes. In fact, one of the primary tools of modern genomics is to predict a gene's role by comparing its sequence to known genes across species. This very principle — that similarity reflects purposeful design and functional patterns — was anticipated by Morris in 1975 and is now routine in biology. Far from being a surprise to creationists, the discovery that genes are conserved, ordered, and functionally significant stands as a striking confirmation of the creationist prediction. Despite the unexpected nature of these findings, most evolutionary interpretations have continued to incorporate both genetic similarities and Hox gene conservation as evidence supporting common ancestry rather than reconsidering prior predictions outside the evolutionary framework. Instead of viewing the data as falsifying earlier predictions, the results have simply been reinterpreted within the existing evolutionary framework. This illustrates a broader pattern in which anomalous or surprising discoveries and failed predictions are accommodated by theoretical adjustment rather than prompting the alternative explanatory YEC model.

Since “design” necessarily implies agency, and agency remains the best inference from the evidence, design is the most compelling conclusion. The strong predictive power of the Young Earth Creation framework serves as the tool that demonstrates why design is not only an inference, but the best explanation.

The Collapse of the Tree of Life

Goal: To reconstruct the “family tree of life” (called a phylogenetic tree), showing how species or genes are related through common ancestry.

Data used: Traditionally, scientists used physical traits (morphology). Today, most phylogenetics relies on DNA, RNA, or protein sequence data and homology.

Phylogenetic tree: A diagram that looks like a branching tree, where:

- Branch points (nodes) = common ancestors.
- Branch lengths = sometimes represent time or amount of genetic change.

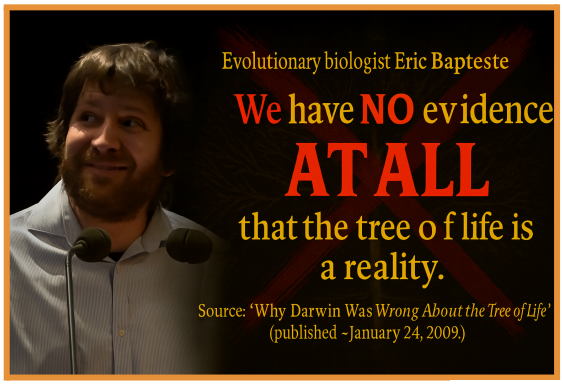
How are trees made? First, investigators choose a gene, or a set of genes, found across multiple organisms. Next, those genes are analyzed to determine their nucleotide sequences, so the gene sequences of various organisms can then be compared. Finally, an evolutionary tree is constructed based upon the principle that the more similar the nucleotide sequence, the more closely related the species. The “tree of life” has long been presented in textbooks as a central framework for understanding evolutionary relationships. This neat branching diagram implies that all organisms descend from common ancestors in a progressively divergent pattern. Yet molecular biology has revealed a far more complex and contradictory reality. Instead of reinforcing the Darwinian tree, genetic data have fractured it into conflicting, often irreconcilable histories.

It is important to recognize at the outset that claims of common ancestry are based upon assumptions rather than direct demonstration. Francis Collins (2006), molecular geneticist, leader of the Human Genome Project, and later Director of the NIH, acknowledged: “*This evidence alone (of genetic similarities between humans and chimps) does not, of course, prove a common ancestor; from a creationist perspective such similarities could simply demonstrate that God used successful design principles over and over again.*” While Collins himself accepted evolution, his admission underscores the philosophical presuppositions underlying the interpretation of similarity as descent. Now that you have learned that genetic similarity was a creationist prediction, these similarities should not only be expected but also evidence for design.

Another shocking admission was published on June 18th 2009, according to research from the University of Pittsburgh and the Buffalo Museum of Science. Reporting in the June 18 edition of the Journal of Biogeography, the researchers reject as "problematic" the popular suggestion, based on DNA analysis, that humans are most closely related to chimpanzees, which they maintain is not supported by fossil evidence. They state: "*There is no theory holding that molecular similarity necessarily implies an evolutionary relationship; ... and molecular data that contradict the idea that genetic similarity denotes relation are often dismissed.*" Physorg.com, "Humans Related to Orangutans, not Chimps, http://www.physorg.com/news164508477.html.

A 2009 *New Scientist* cover story entitled *Why Darwin Was Wrong About the Tree of Life* explained that expectations of a universal tree collapsed as soon as sequencing expanded beyond RNA to DNA (Lawton 2009). Genes often contradicted one another. RNA might suggest species A is closer to B than to C, while DNA would suggest the reverse. Michael Syvanen's attempt to build a tree from 2,000 genes across diverse animals likewise failed: "*He failed. The problem was that different genes told contradictory evolutionary stories... Roughly 50 per cent of [the sea squirt's] genes have one evolutionary history and 50 per cent another*" (Syvanen 2009). Syvanen admitted bluntly: "*We've just annihilated the tree of life.*"

This is not an isolated phenomenon. As W. Ford Doolittle (1999) concluded: "Molecular phylogenists will have failed to find the 'true tree,' not because their methods are inadequate or because they have chosen the wrong genes, but because the history of life cannot properly be represented as a tree." Carl Woese (1998), pioneer of molecular systematics, concurred: "*Phylogenetic incongruities can be seen everywhere in the universal tree, from its root to the major branchings within and among the various taxa.*" Evolutionary biologist Eric Bapteste later summarized: "*We have NO evidence AT ALL that the tree of life is a reality.*" (New Scientist, January 24, 2009 *Image 4).*



(Image 4)

"studies of hominids, pines, cichlids, finches, grasshoppers and fruit flies have all detected genealogical discordance so widespread that no single tree topology predominates" - James H. Degnan 2009

Conflicts extend across levels of analysis. Morphology-based trees often contradict molecular ones. Liliana Dávalos et al. (2012) reported: "Incongruence between phylogenies derived from morphological versus molecular analyses, and between trees based on different subsets of molecular sequences has become pervasive as datasets have expanded rapidly in both characters and species." Even highly sequenced proteins fail to resolve the problem.

Even Cytochrome c trees rarely match the standard evolutionary pattern, while cytochrome b yields what Michael Lee (1999) called an "absurd phylogeny," placing cats and whales within primates. Leonard Brand (1997) likewise reported anomalies such as human cytochrome c being closer to kangaroos than to horses.

The contradictions are so deep that entire sections of the supposed tree reduce to "**bushes**." Rokas & Carroll (2006) admitted that "the recurring discovery of persistently unresolved clades (bushes) should force a re-evaluation of several widely held assumptions of molecular systematics." In the same vein, Trisha Gura (2000) observed in Nature: "Evolutionary trees constructed by studying biological molecules often don't resemble those drawn up from morphology." Michael R. Rose, an evolutionary biologist, conceded: "The tree of life is being politely buried."

Ingo Ebersberger et al, in the journal article titled: Mapping Human Genetic Ancestry, 2007, state: *"Thus, in two-thirds of the cases [trees], a genealogy results in which humans and chimpanzees are not each other's closest genetic relatives. The corresponding genealogies are incongruent with the species tree. In concordance with the experimental evidences, this implies that there is no such thing as a unique evolutionary history of the human genome. Rather, it resembles a patchwork of individual regions following their own genealogy."*

Even more striking are cases where highly similar, functional genes occur in organisms with no plausible shared ancestor. A recent report in Science noted: "*Two active genes from the silkmaking amphipod legs are found in silkworms... leads us to believe they are using very similar genes as the moths... But why and how those similar genes arose—it's unlikely they date back to a common ancestor—is what keeps me up at night.*" (Science, 2024). Such findings fit a design-based paradigm but remain anomalous under a strict evolutionary paradigm.

Both "Tree Thinking" and "Evolution 101" have failed miserably at this. Of the 49 tree of life drawings in Evolution 101, not a single tree (0%) includes the name of a common ancestor. That's right, and Tree Thinking, an Introduction to Phylogenetic Biology admits "Our knowledge of the molecular process is not good enough to definitively rule out independent origins." But that is not all it says, look at what they begrudgingly admit.., *"With the ever-increasing volumes of data, the incongruence between trees has become pervasive."*

The situation is now openly admitted by evolutionary biologists. Nicolas Galtier et al. (2008) summarized: "Incongruence between gene trees is the main challenge faced by phylogeneticists in the genomic era." Pat Willmer (2013) put it more bluntly: "*In point of fact, there exists no such thing as the 'traditional textbook phylogeny.' A diversity of different schemes can be found.*" What about histones, since these are proteins within DNA that are found within all living organisms, from bacteria to mankind?

Clearly, those would make a good way to test ancestry, right? Maybe even a molecular clock, right? Well, guess what? They tried that as well, and the results came back in favor of YEC regarding the fast rate, and the tree that emerged did not show the evolution of life they needed. "Histones are not used for molecular clocks because they…do not confirm preconceived ideas about how the Precambrian tree of life ought to look like." (Stephen C. Meyer 2013) "*…small differences between histones yield an extremely recent divergence, contrary to other studies.*" Meaning the mutation rate did not match the evolutionary timeline. Therefore, evolutionary biologists now exclude histones from consideration since they do not fit the narrative. The weight of evidence now compels us to acknowledge that the tree of life, long presented as an unquestionable fact, lies in ruins.

As New Scientist concluded: "*For a long time the holy grail was to build a tree of life … But today the project lies in tatters, torn to pieces by an onslaught of negative evidence*" (Bapteste 2009). The continued reliance on ad hoc explanations—horizontal gene transfer, convergent evolution, flawed sampling—reveals an unwillingness to reconsider the central assumption of universal common ancestry. In the end, if the data themselves repeatedly defy key predictions and the treelike model, then intellectual honesty requires us to admit that the "tree of life" is not a fact of nature, but at best a metaphor—one that has outlived its usefulness.

Evolutionists currently had to remove homology and then cherry pick single genes to build a tree of life that portrays what they believe evolutionary history should look like. That is what they have to resort to since everything else has failed them and contradicts the data. More force fitting data while ignoring the obvious picture and vast majority of evidence and data-neglecting the rule.

On the creationists' side of this, we have Frank Lewis Marsh (1899–1992) – Seventh-day Adventist biologist, "Father of Baraminology" who first formalized the term baramin ("created kind") in his book Fundamental Biology (1941). Predicted that genetic boundaries exist: organisms may vary widely within a baramin but will not cross into another. This was further elaborated on the genetic side by Henry M. Morris, quoted earlier, who extended Marsh's predictions in Scientific Creationism (1974) and The Genesis Flood (1961, with John Whitcomb). Where he predicted that:

1. Genetic similarity would be found within kinds but not across them.
2. Taxonomic overlap across kinds would not be possible — "variation is real, but only within created limits."

> Quote: "*True species are genetically isolated units; variation and hybridization can occur only within the bounds of created kinds.*" *(Scientific Creationism, 1974, p. 84).*

What has only been recently discovered? The on par results again with creationists' predictions, we have discovered in 2018 regarding genetic boundaries. It was at this time that scientists had the ability to sequence a part of DNA called the Cytochrome C Oxidase subunit I protein (COI) very cheaply. Around 2002, Canadian molecular biologist Paul Hebert, who coined the term "DNA barcode," figured out a way to identify species by analysing the COI gene. In analysing the barcodes across 100,000 species, the researchers found a telltale sign showing that almost all the animals emerged about the same time as humans. *"Namely that the extant population, no matter what its current size or similarity to fossils of any age, has expanded from mitochondrial uniformity within the past 200,000 years"* (M. Y. Stoeckle and D. S. Thaler 2018)

In an article titled: Sweeping gene survey reveals new facets of evolution by Marlowe Hood, he interviewed both leading scientists Thaler and Stoeckle who admitted this: *"yet-another unexpected finding from the study- species have very clear genetic boundaries, and there's nothing much in between. "If individuals are stars, then species are galaxies," said Thaler. "They are compact clusters in the vastness of empty sequence space." "The absence of "in-between" species is something that also perplexed Darwin, he said."*

So we see two things stand out. Genetic boundaries and low genetic diversity that is equal in all life. This is best explained from the YEC model since mutation rates are fast and there is still low genetic diversity, this means not a lot of time has passed. The 200,000-year date that they comment on is the assumed most recent global bottleneck in the evolutionary timeline. It has no evidence for it, as we can from multiple studies that have gone looking for this mysterious bottleneck in the fossil record and came back empty-handed. Timothy D. Weaver in the Journal of Human Evolution Volume 63, Issue 1, Pages 121-126 titled Did a discrete event 200,000-100,000 years ago produce modern humans? Quotes Bräuer, 2008: *"nothing out of the ordinary happened 200,000-100,000 years ago in Africa."* Science writer Michael Marshall further elaborates and confirms, stating*: "Second, there is no trace in the geological record of any global event in the last 200,000 years. Any event that slashed populations that significantly would surely have led to a noticeable spike in the extinction rate, and there isn't one."*

We have clear evidence that a genetic bottleneck for all life on Earth occurred, it was catastrophic and global. Nothing in the evolutionary interpretation of the fossil record exists for a global bottleneck 200,000 years ago or even close to this timeframe. Therefore, we are left with the genetic evidence that tells us a very different story from the evolutionary one, and one that points to Noah's global flood. Now, take away the phylogenetic assumption given regarding how often a nucleotide changes, and we can answer the true question of "when did this bottleneck occur"? Further elaboration on the timeline will be made in a subsequent chapter of mutation rates where we unequivocally confirm this mythical 200,000 year timeline which was conveniently made up as a rescue device to try to explain low genetic diversity and nothing more.

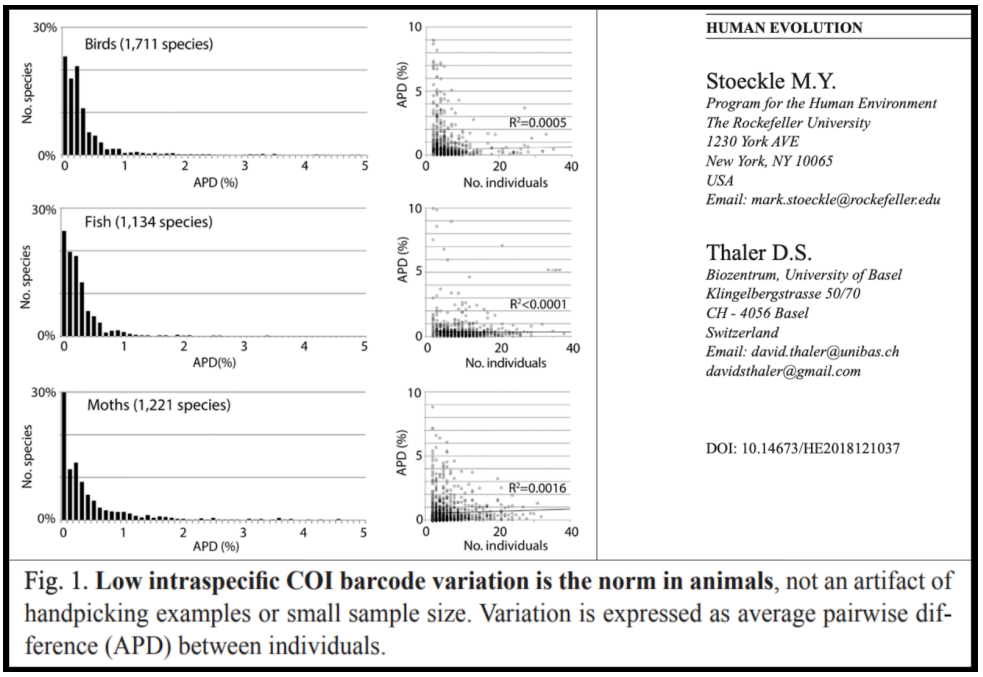


Figure 1 from the 2018 Rockefeller University study showing birds, fish and moths low genetic diversity inside the COI gene fragment.

In the interview with Marlowe Hood Thaler admitted that: "*This conclusion is very surprising, and I fought against it as hard as I could,*" Thaler told AFP." Hood, M. (2018, May 28). Sweeping gene survey reveals new facets of evolution.

DNA barcoding demonstrates that new species can retain ancestral polymorphisms, which has three important implications.

(1) All life is relatively young and remains genetically identifiable both within and between species.

(2) Genetic boundaries help us identify what organisms were present on Noah's ark and survived the post-Flood bottleneck, since variants that arose by mutation before speciation can be traced back to their ancestral source.

(3) The persistence of ancestral polymorphisms complicates the interpretation of evolutionary molecular interpretations and can lead to incorrect phylogenetic inferences. Importantly, this does not undermine the utility of DNA barcoding. Be sure to see my study titled. One Species, Many Names: Mitochondrial Evidence Unites Humans, Neanderthals, Denisovans, and Heidelbergensis. Where I use DNA barcoding to track hominins backwards in time, converging on the modern day consensus sequence. Both confirming YEC predictions, and delineating true genetic relationships. For example, in the study I show that Neanderthals, Denisovans, and modern humans are not hybrids of different species or a subspecies, but rather members of the same species who lived contemporaneously and diverged after the bottleneck—retaining common ancestral polymorphisms in their genomes.

These genetic boundaries allow us to track all related species back to a consensus sequence where they all diverge from after a bottleneck, showing us what species are related through convergence and answering what a biblical "kind" is. For example, in a study where I took all felines and ran their sequences back in time, I converged on two independent consensus sequences. One for large cats and one for small cats. This means that all large cats we see today descended from a single large cat species after the last bottleneck, and the same for all small cat species. A clear contrast to the evolutionary assumption that big cats arose and evolved starting 2 - 4 million years ago, based on fossils. For more details, see: When Barcodes Connect: Mitochondrial DNA Barcoding of Felidae Indicates Two Ancestral Lineages? (Nailor, M. 2025). "*DNA barcoding will deliver species-level resolution in 95% to 97% of all cases* (Hebert et al., 2004b; Janzen et al., 2005; Ward et al., 2005*).*" This makes it extremely accurate and the best region for testing ever discovered.

The YEC model predicts that genetic diversity for mitochondria originated recently, and the fast rates of mutation seen throughout the mtDNA are clocks we can use to date historical events and life itself. These high rates of mutation allow for rapid speciation and hybridization. These rapid radiation and speciation events are also observed and well documented in everything from birds, plants, fish, and reptiles (Fan Han et al., 2018; Joanna M. Buswell et al., 2010; Y. E. Stuart et al,. 2014; Fang Zhu et al., 2013; Claire Brandenburger et al., 2019; Hudson CM et al,. 2020), contrary to Darwinian evolution known as gradualism which was predicted to take tens of thousands of years or more.

Rather, the new observable data confirm predictions of the YEC model. All of these published discoveries contradict key predictions of neutral theory. The fast rates of change in the COI gene are another evolutionary falsification by showing that the assumed calibrated rate of 1%/million years that is calibrated from fossils or older vicariant events (Crandall et al. 2012) is a presupposition based on evolutionary assumptions and nothing more.

Evolutionary Neutral theory still predicts a wide range of variations in all life on Earth. With higher levels of variation in organisms that reproduce more rapidly, as the rate of diversity recovery after a selective sweep or genetic bottleneck is proportional to generation time *(time to equilibrium variation is N generations; Hartl DL, Clark AG 2007, Bedford T, Cobey S, Pascual M 2011)*. In **contrast** to this prediction, is the YEC prediction that a recent global bottleneck took place and all life would reflect the same level of variation which has been discovered through the widespread effectiveness of DNA barcoding which reflects similarly low levels of intraspecific variation across the diversity of all animal life, including insects and vertebrates that differ 100-fold in generation time (Hajibabaei et al., 2006; Ward et al., 2005).

The YEC view solves Lewontin's Paradox and adds another accurate prediction under its proverbial belt. This paradox refers to the observation that genetic diversity across different species varies much less than expected compared to the vast differences in their population sizes, meaning that species with vastly different population numbers exhibit similar levels of genetic diversity, which contradicts the prediction that larger populations should have more genetic variation; this paradox was first described by Richard Lewontin in 1974. This paradox still exists, as well as the Genetic Equidistance Paradox, which is the observation that all life shows a surprising lack of genetic diversity and appears roughly equidistant when compared across wide evolutionary gaps. Discovered in the 1960s when scientists compared protein/DNA sequences across very different organisms. They found that, surprisingly, different species were about equally distant from each other genetically, regardless of how different they looked or how long ago they supposedly diverged.

An example of this is humans, which are about as genetically different from frogs as they are from fish, when looking at certain molecular sequences. Under straightforward evolutionary expectations, you'd predict that more ancient lineages (like fish vs. humans) should show far greater genetic divergence than more recent ones (like primates vs. humans). Instead, the differences appeared to "*plateau*" — suggesting a limit to measurable genetic diversity across life. This is still a problem, and what Stoeckle tells Marlowe Hood in an interview for phys.org: *"It is textbook biology, for example, that species with large, far-flung populations—think ants, rats, humans—will become more genetically diverse over time.*
But is that true?

"The answer is no," said Stoeckle, lead author of the study, published in the journal Human Evolution. For the planet's 7.6 billion people, 500 million house sparrows, or 100,000 sandpipers, genetic diversity "is about the same," he told AFP.

What solves these paradoxes? The YEC view is that there has been a recent bottleneck with very little time for mutations to accrue large-scale genetic diversity. The "Genetic Equidistance Paradox" is an easy argument to make as a YEC since the pattern we see does not fit the evolutionary timeline, but does fit with a model of created design, limited variation, and recent ancestry, with all life going through a global bottleneck.

The real question those who hold to common descent need to ask is: if the theory of evolution is true, why does all life have such low levels of genetic diversity? Since most of the differences that separate us all have arisen on this side of the bottleneck, where are the missing millions of years of genetic diversity before the bottleneck? This again is also best answered from the YEC view of recent creation and not deep time divergences via evolution. Those missing mutations are another major problem for evolution, which you will never hear brought up. We will get more into mutation saturation later, which confirms this paradox as well.

This genetic evidence matches the taxonomic boundary paradox that plagues the evolutionary model but aligns with the genetic and homology data predicted by YEC. One such example is that of baraminology discordance, which allows us to test fossils that have no reliable DNA.

Kurt Wise in 1990, a Harvard-trained paleontologist, predicted we would find boundaries as well between kinds. This prediction was taken by Todd Charles Wood, PhD — an American biologist and researcher known for formalizing statistical methods in baraminology (like BDIST, baraminic distance correlation).

- *He has repeatedly emphasized that* ***if baraminology is valid, then taxonomy should reveal "discontinuity" between created kinds but "continuity" (variation and clustering) within them.***

Examples of predictions he's made

- *Sharp taxonomic boundaries (discontinuities).*
- *In multiple papers and presentations (early 2000s onward), Wood has predicted that as we study more genomes and apply statistical clustering methods, we will continue to find sharp discontinuities between baramins (created kinds), not smooth transitions across all life.*
- *For example, humans will always cluster with hominins such as neanderthal, Homo erectus, Denisovan, and Heidelbergensis. Wolves and coyotes will cluster with dogs, etc., with significant gaps between them.*

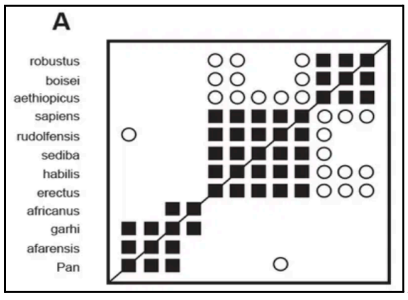


Figure 2. Baraminological distance scale chart.

- ***Consistency across data types***
- *He has predicted that these same boundaries should appear whether you use morphology (physical traits) or molecular data (genomes).*
- *In other words, different types of evidence should independently confirm the same created kinds.*

With the new genetic DNA barcoding data, we can now confirm such predictions. With one of these goals being able to determine a holobaramin, aka an original created kind.

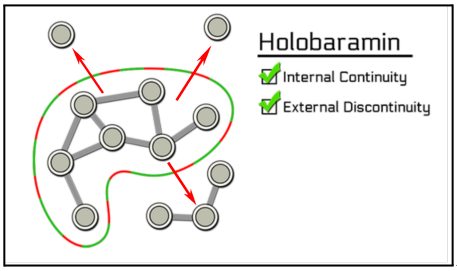


Image 5.

- *Prediction against "gradualism"*

- Wood has argued that, unlike evolutionary systematics (which expects continuity and gradual transitions), baraminology will show and continue to show that biological diversity forms distinct clusters.

- He systematized baraminology with statistical tools ("discontinuity systematics").

- He directly predicted that baraminology will reveal sharp discontinuities (gaps) between created kinds, but clusters of variation within kinds.

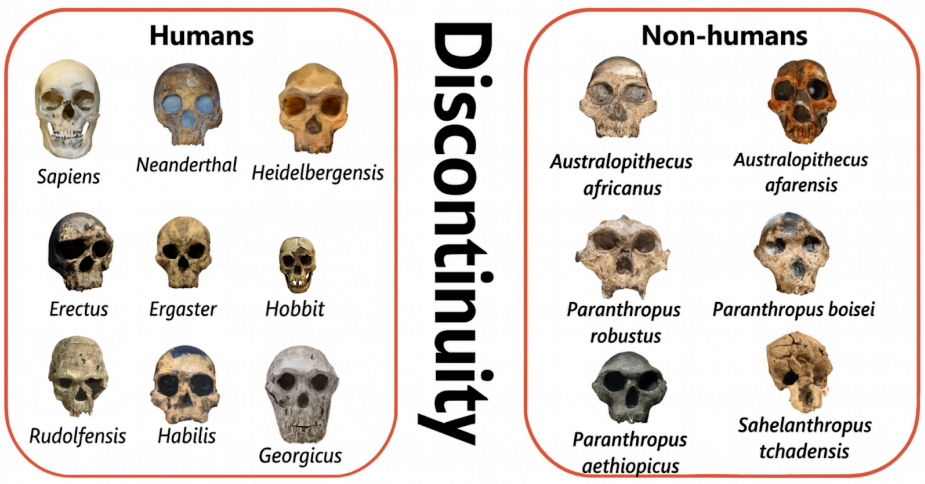


Image 6.

- Todd C. Wood, 2006, wrote that baraminological methods predict sharp discontinuities in biology, not gradual continua.

- ***Todd C. Wood, 2010****, The BioLogos Forum: He stated that creationism makes* ***"real scientific predictions"*** *— one of which is that taxonomy will reveal* ***discontinuity between kinds*** *and* ***clusters of variation within kinds****.*

- ***Journal of Creation / ICC proceedings****: He has made repeated references to the prediction that baraminological analyses will consistently recover distinct groups (baramins), regardless of what data is analyzed.*

We repeatedly observe (i) pervasive incongruence among gene trees and between genes and morphology, (ii) bushes and unresolved clades where a branching hierarchy was expected, and (iii) clear genetic boundaries coupled with low, tightly binned intraspecific variation across animal life. These patterns persist across loci, methods, and taxonomic scales. Far from being an occasional nuisance to be patched by post-hoc explanations, they form the dominant signal in the data. Discontinuity between kinds: Independent datasets repeatedly recover sharp gaps that resist smoothing into a universal tree. Continuity within kinds: Species within recognized clusters show abundant, structured, but bounded variation—including shared ancestral polymorphisms traceable through time. Cross-data agreement: Morphological and molecular evidence, when analyzed with clustering rather than forced bifurcation, converge on the same groupings. Barcoding adds a fourth reinforcement: synchronous, low intraspecific diversity across the animal kingdom. That finding is hard to square with neutral theory expectations (diversity scaling with population size and time since sweep), but is exactly what one expects after a recent, global bottleneck followed by limited time for divergence.

To be clear, none of this denies variation, adaptation, or rapid radiation—indeed, these are essential features of the post-bottleneck world documented across birds, fish, plants, and reptiles. The point is scope and structure: variation appears fast, directional, and bounded, producing distinct clusters rather than an indefinitely graded continuum.

Henry Gee Paleontologist and evolutionary biologist and Senior Editor of Nature states: *"No fossil is buried with its birth certificate... To take a line of fossils and claim that they represent a lineage is not a scientific hypothesis that can be tested, but an assertion that carries the same validity as a bedtime story - amusing, perhaps even instructive, but not scientific."* Source: In Search of Deep Time: Beyond the Fossil Record to a New History of Life (Ithaca: Cornell Univ. Press, 1999), p. 96

From a design-based/YEC framework, these results were *predicted*. Creationist biosystematics (baraminology) anticipated **sharp discontinuities between "created kinds"** with **substantial but bounded variation within kinds**.

- ***Empirical signal:*** *Widespread gene-tree incongruence, unresolved "bushes," of life and cross-marker clustering dominate all known datasets.*

- ***Boundaries:*** *Species form compact genetic clusters with* ***clear gaps*** *and* ***little in-between****, consistent with designed limits and a recent common bottleneck.*

- ***Low diversity:*** *Uniformly* ***low intraspecific barcode diversity*** *across animals—independent of population size or generation time—fits* ***recent*** *bottleneck expectations and strains neutral-theory scaling.*

- ***Predictions met:*** *Longstanding YEC/baraminology predictions (discontinuity between kinds; continuity within kinds; agreement across data types) are borne out by modern datasets.*

- ***Next step:*** *Later in this study, it quantifies* ***observed germline mutation rates*** *and shows how they anchor a shorter chronology that coherently explains the clustering, the boundaries, and the paucity of accumulated diversity while simultaneously landing on the Biblical timelines.*

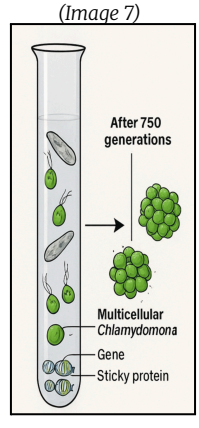
So we have taxonomic boundaries, morphological boundaries, and genetic boundaries. None of these were expected by the theory of evolution; as a matter of fact, the exact opposite was expected and predicted. It was YEC who predicted we would find such things, and confirmation has resulted over the decades. In sum, after more than half a century of molecular systematics, the strongest, most repeatable patterns are clusters and limits, not a single, universal bifurcating tree. A model that begins with created kinds, a recent global bottleneck, and fast single-generation rebounded post-bottleneck diversification provides a tighter, more predictive fit to the data—and sets the stage for the mutation-rate analysis that follows later in this paper.

> *"My attempts to demonstrate evolution by an experiment carried on for more than 40 years have completely failed... It is not even possible to make a caricature of an evolution out of paleobiological facts... The idea of evolution rests on pure belief."* Dr. Nils Heribert-Nilsson, noted Swedish botanist and geneticist, of Lund University

Multicellular Evolution

One of the central claims of evolutionary theory is that multicellular organisms arose from single-celled life through gradual innovations over billions of years (Maynard Smith & Szathmáry, 1995). This transition is considered crucial, since multicellularity allows for specialized tissues and greater biological diversity. Laboratory experiments with green algae have been presented as evidence for this process. For example, Chlamydomonas reinhardtii—a single-celled alga—was placed in test tubes alongside a predator, Paramecium tetraurelia. After 750 generations, some Chlamydomonas began forming multicellular clusters large enough to escape predation (Herron et al., 2019) Image 9. Evolutionists argue that this demonstrates an early step toward multicellularity, echoing supposed evolutionary transitions between unicellular algae and colonial forms like Volvox (Arakaki et al., 2013).

(Image 7)



However, genetic evidence indicates that these results do not reflect the origin of new biological information. Chlamydomonas and Volvox share many of the same genes, including those necessary for multicellularity, such as those coding for sticky proteins that allow cells to adhere (Prochnik et al., 2010). The difference is largely in gene expression and copy number, not in the appearance of novel genes. Indeed, researchers found that up to 20% of Chlamydomonas genes changed expression under predation pressure (Herron, 2016). This demonstrates that the algae already possessed the built-in capacity to form cell clusters, which was merely "switched on" by environmental cues. No new genes or complex structures arose in these experiments.

Thus, claims that these experiments reproduce the evolutionary origin of multicellularity are misleading. Instead, the results highlight the remarkable adaptability of organisms within their created limits. The observed shifts are better explained as activation of latent genetic potential or even loss of complexity, rather than the stepwise emergence of new multicellular programs. As Genesis 1 affirms, life was created according to its kinds, with built-in capacity for variation but not transformation into fundamentally new forms. In this light, the evidence supports design and creation rather than microbes-to-man evolution.

Endosymbiosis and the Origin of Eukaryotes

Another core evolutionary claim concerns the supposed origin of eukaryotic cells through the engulfment of one prokaryote by another, popularly known as the **endosymbiotic theory** (Margulis, 1970). According to this view, mitochondria and chloroplasts were once free-living bacteria that became permanently incorporated into a host cell, giving rise to the first eukaryote. The model has been widely heralded as a landmark in evolutionary biology, often presented with an air of near certainty in textbooks and popular science writing (Sagan, 1967; Martin et al., 2015).

Yet despite its prominence, the central process it invokes has **never been observed**. No experiment has ever documented a prokaryote engulfing another prokaryote and transforming into a fully integrated eukaryotic cell with stably inherited organelles and cooperative genomes. Nor has such a transformation been observed in nature. Laboratory demonstrations of transient symbioses — for instance, amoebae temporarily harboring bacteria (Nowack & Melkonian, 2010) — fall far short of the wholesale genomic integration required by the theory. At most, these examples demonstrate that microbes can live in close association, but they do not replicate the origin of mitochondria or chloroplasts.

Even the strongest evidence cited for endosymbiosis is inferential rather than direct. Similarities between mitochondrial and bacterial ribosomes, or between chloroplast and cyanobacterial DNA, are taken as proof of descent (Gray, 2012). Yet such similarities can equally be interpreted as evidence of **common design**: functional systems employing similar molecular machinery because they fulfill similar roles. Moreover, the reductive nature of organellar genomes — far smaller than their bacterial counterparts — underscores a major explanatory gap: if mitochondria and chloroplasts truly began as independent bacteria, what mechanisms drove the massive and coordinated transfer of thousands of genes to the host nucleus, all while preserving viability and regulatory integration? Evolutionary accounts of this "gene transfer" remain speculative (Martin & Müller, 1998).

The theory also relies on events that are, by definition, **unrepeatable and outside observational science**. Evolutionary biologists invoke a unique, exceedingly rare fusion event in the deep past, but such ad hoc explanations cannot be tested, repeated, or falsified. Lynn Margulis herself, the chief proponent of the theory, later admitted that the evidence remained circumstantial and that key steps had not been experimentally demonstrated (Margulis, 1993). More recent reviews acknowledge that critical questions remain unresolved — including how an archaeal host could have engulfed a bacterium in the first place, since prokaryotes lack the phagocytic machinery of modern eukaryotes (Cavalier-Smith, 2002; Booth & Doolittle, 2015).

From a creationist perspective, this absence of empirical demonstration is decisive. The endosymbiotic model is a story constructed around similarities, but one that fails the Popperian standard of falsifiability: the central mechanism — engulfment and stable genomic integration — has never been observed, cannot be reproduced, and therefore remains outside the bounds of operational science. Instead, the data are better explained as evidence of design: mitochondria and chloroplasts were fully functional organelles from the beginning, embedded within cells as part of an integrated system. Complex, interdependent features such as dual-genome coordination, protein import machinery, and finely tuned regulatory networks suggest foresight and purposeful design rather than an accidental merger of independent organisms.

In short, the confident claims surrounding the endosymbiotic theory mask the fact that **there is no direct observational evidence for this process**. Rather than confirming evolutionary predictions, the data reinforce the view that eukaryotic complexity was present from the start.

Then, in 1977, Carl Woese): Used ribosomal RNA sequences to map relationships across life, discovering the three domains (Bacteria, Archaea, Eukarya). This was a massive milestone: clear genetic similarity forming a nested hierarchy at the molecular level,

confirming the second YEC prediction by Morris about there being similar underlying genetic properties, reflecting a unified pattern of functional design. Then, in the 1980s and onward, Widespread DNA sequencing confirmed that genetic data produce the same nested hierarchical patterns long noted by creationist taxonomist Carl Linnaeus, and homologist Sir Patrick Owen, who stated: *'that ideal original or fundamental pattern on which a natural group of animals or system of organs has been constructed. And to modifications of which the various of such animals or organs may be referred."*

The analogy between human design and biological structures has a long history within design-based explanations of life's origins. As early as 1802, William Paley—often considered a pioneer of natural theology—compared living organisms to human-engineered machines. He argued that recurring structural patterns across different species could be explained in a manner analogous to how engineers adapt a common design for multiple purposes. He wrote, *"Whenever we find a general plan pursued, yet with such variations in it as are, in each case, required by the particular exigency for the subject... we possess, in such a plan and such adaptation...the strongest evidence for intelligence and design ... .Arkwright's mill was invented for spinning cotton. We see...such modification of the original principle, such variety of the same plan... to observe it in different applications [for spinning wool, flax, and hemp]... .Very much of this reasoning is applicable to what has been called comparative anatomy. In their general economy, in the outlines of the plan, in the construction as well as offices of their principal parts."* (Paley, W. 1802).

Proponents of evolutionary theory have subsequently adopted this concept, reframed it within an evolutionary framework, and employed it in support of their arguments. However, the idea itself originated within the Young Earth Creationists, as did much of the other evidence now referenced.

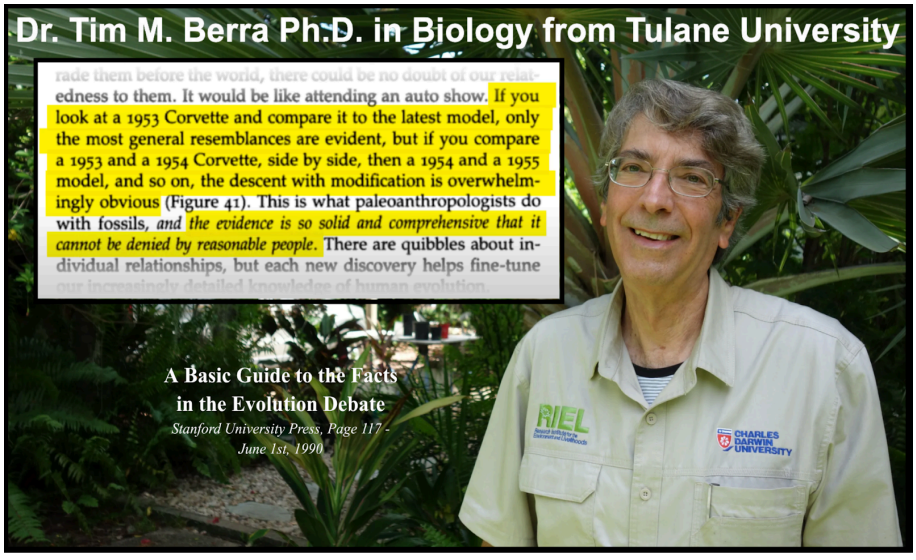


Image 8.

It is common for modern-day prominent evolutionary theorists to have historically rejected common design explanations after Darwin, even when such predictions were subsequently validated by genetic and anatomical evidence. During Darwin's time, the rationale was often framed in theological terms. For example, Darwin himself used his new popularity to press doubt on the masses by saying things like *"Nothing can be more hopeless than to attempt to explain this similarity of pattern in members of the same class, by utility or by the doctrine of final causes. It is at least as noble a conception to believe that it has pleased the Creator to construct all the animals and plants in each great class on a uniform plan, than to believe that he required a separate act of creation to bring each into existence." (Origin of Species, 6th ed., 1872, Chapter XIV: Recapitulation and Conclusion, p. 429*). All the while at the same time plagiarizing and copying creationists content and ideas. Darwin's formulation of the popular phrase; *"similarity due to common ancestry"* contrasted with earlier creationist perspectives, notably those of Professor Sir Richard Owen, who had advanced the concepts of homology and archetype theory and described such similarities as *"due to common design."* Historians of science have also noted that elements of Darwin's natural selection theory overlapped with, and may have been influenced by, prior work by Edward Blyth, Patrick Matthew, Alfred Russel Wallace, and others (see [citations]).

Ph.D. Rhawn Gabriel Joseph points out that on June 8, 1858, Darwin received a letter from Alfred Russel Wallace, accompanied by a 12 page summary of Wallace's ideas on natural selection. *"Darwin immediately abandoned the study of barnacles and began feverishly working on a book, a synthesis of the words of Blyth, Wells, Pritchard, Lawrence, Naudin, and Buffon: On the Origin of Species by Means of Natural Selection which he published in November of 1859, almost 18 months after receiving the paper by Wallace.' Darwin later claimed to have recently arrived at identical conclusions, and thus claimed Wallace's theory as his own."* [Rhawn, J., pp. 223–226, 2000]

Between 1835 and 1837, 24 years before Darwin's *On the Origin of Species*, Edward Blyth published a series of articles in *The Magazine of Natural History* in which he offered detailed observations of bird variation (particularly among Indian species) and commentary on selective breeding practices. These contributions significantly enriched the scientific "pool of knowledge" and predated Darwin's *On the Origin of Species* by nearly 24 years (Darwin Online, 2023; Current Conservation, 2021; National Center for Science Education, 2018; Encyclopedia.com, 2019). Although Blyth's views differed in emphasis—he viewed natural selection more as a mechanism for maintaining species integrity for survival rather than generating entirely new forms—the empirical observations in his work align more closely with modern data than Darwin's later theoretical reframing.

Patrick Matthew's actually *"... anticipated Darwin's main conclusions by twenty-eight years, yet he thought them so little important that he published them as an appendix to his book ... and did not feel the need to give substance to them by continuous work.*

Darwin's incessant application, on the other hand, makes one think that he had found in evolution and its related concepts, not merely a scientific theory about the world, but a vocation ... .'[De Beer, G., p. 11, 1969]

Stephen J. Gould even admits that Darwin was influenced by many people, and could have developed his ideas from them **[**Macrone, M., p. 150, 1994**]**. Gould notes that: *'Matthew, still alive and vigorously kicking when Darwin published the Origin, wrote to express his frustration at Darwin's non-citation*' [Gould, S.J., 1979; p. 38.].

Evolution was, from its conception, nothing more than a wild idea that caught the attention of atheists who used it for their agenda to attack the popular narrative at the time, which was Biblical creation. It really started with Charles Lyell, a lawyer who was highly influenced by Jean-Baptiste Lamarck, a French naturalist, soldier-turned-botanist, and later zoologist. He believed organisms progress from simple to more complex forms over long periods, driven by some internal "force of life." By Darwin's time, Lamarck's mechanism of "inheritance of acquired characteristics" was largely rejected, since it didn't fit observed patterns of heredity. But Lyell saw its power and even stated in a 1827 letter that Lyell wrote to Gideon Mantell. In that correspondence, Lyell expressed enthusiastic engagement with Lamarck's ideas in this memorable way:

"***I devoured Lamarck... his theories delighted me****... I am glad that he has been courageous enough and logical enough to admit that his argument,* ***if pushed as far as it must go, if worth anything, would prove that men may have come from the Ourang-Outang****. But after all, what changes species may really undergo!... That the Earth is quite as old as he supposes, has long been my creed...*" (*Life, Letters and Journals of Sir Charles Lyell, Bart*, Vol. I, p. 168)

This was the fuel for the fire, so to speak, Lyell's Principles of Geology so reshaped Darwin's worldview that Darwin later said he often 'viewed the natural world through Lyell's eyes.' Lyell was literally inventing his own history and implementing Lamarck's ideas... The trajectory of his influence on Darwin is evident."

Darwin wrote that when he viewed geological features, he did so partly "through Lyell's eyes":

> "*The Principles ... altered the whole tone of one's mind and thence when seeing a thing never seen by Lyell, one yet saw it partially through his eyes.*" (Darwin, 1838; quoted in Vaughan, 2011)

Lyell even admitted in his journal that was found after his death that his goal was to "rid the sciences of Moses". He expressed this intention in a letter to George Poulett Scrope:

> *"I am sure you may get into [Quarterly Review] what will free the science from Moses ... if ever the Mosaic geology could be set down without giving offence, it would be in an historical sketch, and you must abstract mine..."*

Lyell's insistence that *"the present is the key to the past"* removed the short, catastrophic timeline and popularized the idea of a long, continuously changing Earth and his *Principles of Geology* was indeed written as an alternative history of the planet. One where he made up the rules and timescale to dupe the world into this idea. The truth is the past is key to the present, not the other way around.

Lyell's work was not merely an exercise in science, but also an attempt to advance a new, secular interpretation of Earth's past by removing God altogether. Through his promotion of uniformitarianism, he influenced Darwin to view geology as evidence of 'deep time' and to reinterpret natural history through what Lyell termed 'my history.' By insisting that 'the present is the key to the past,' Lyell dismissed the shorter, catastrophic timescale in favor of a mythical vision of a continuously stable Earth. *Principles of Geology* thus functioned as an alternative narrative of Earth's history—one constructed on Lyell's own assumptions about rules and timescales. Yet, we argue that it is "the past that is the key to the present", rather than "the present serving as the key to the past."

BENEFICAL MUTATIONS

Natural selection, by itself, does not introduce new genetic variants; meaning Natural selection takes evolution nowhere, on its own - rather, it acts upon variation already present in a population by increasing or decreasing the frequency of specific alleles. The real new variation in the evolutionary models requires mutation, which provides the raw material upon which selection may act.

As stated by Evolutionary Naturalist Jean-Baptiste Lamarck, *"Nature, by its own means, has successively produced all species of animals, beginning with the simplest and ending with the most complex."* However, empirical studies raise nothing but questions and doubts about the capacity of mutations to generate entirely new novel anatomical structures in the linear, branching manner represented in textbook diagrams of evolution.

For example, experimental work focusing on Hox genes—master regulators of body-plan development—has demonstrated that mutational changes can alter or disrupt existing developmental pathways, but such alterations have not been observed to produce entirely new, functional anatomical features as expected and required to validate the evolutionary narrative. Instead, the results frequently involve loss of function, duplications, or deformations, consistent with modification of existing genetic information rather than the origination of novel structures (see Carroll, 2005; Carroll et al., 2013). To date, we have the entire genome sequenced of...

- Fruit fly (*Drosophila melanogaster*) – genome completed in **2000** (published in *Science*, part of a big international collaboration with Celera Genomics).

- Nematode worm (*Caenorhabditis elegans*) – first **multicellular organism** to have its genome sequenced, completed in **1998** (published in *Science*).

- House mouse (*Mus musculus*) – genome draft completed in **2002** (published in *Nature*; it was the second mammalian genome after humans).

- **Yeast** (*Saccharomyces cerevisiae*) – first eukaryote to have its genome fully sequenced (1996).

- **Plants** – *Arabidopsis thaliana* (a small flowering plant, model for genetics) completed in 2000.

Experimental manipulations of Hox gene regions in the above organisms are often presented as demonstrations of evolutionary mechanisms. Yet, the outcomes of these targeted alterations consistently result in developmental abnormalities, not the emergence of genuinely novel anatomical structures. For instance, forced mutations in the Hox gene in fruit flies producing additional wings or legs do not represent **new** traits but rather the misexpression of pre-existing structures.

In such cases, the extra wings were non-functional and impaired flight, while legs that developed in place of antennae were malformed and non-adaptive (Lewis, 1978; Carroll, 2005). These results highlight a key limitation: mutations in Hox regulatory regions can reorganize or duplicate existing features but have not been shown to generate entirely new, functional traits. Thus, while these experiments underscore the pivotal role of Hox genes in developmental patterning, they stop short of providing direct evidence for the evolutionary origin of novel new body parts slowly arising.

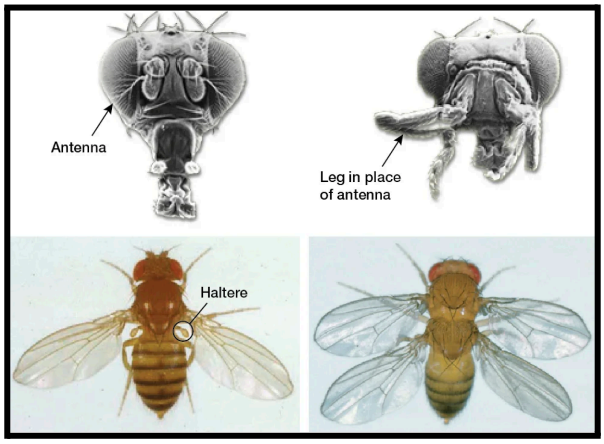


Figure 3 shows (top) a leg growing out of the face where an antenna belongs and (bottom) shows an extra set of wings growing where halteres should be. Halteres are special sensory organs at the base that detect changes in body position and rotation. Halteres act like gyroscopes and help the fly balance as it flies.

Reported cases of so-called 'beneficial mutations' do not demonstrate the origin of new novel anatomical features. Rather, they involve altered expression of existing traits—often accompanied by trade-offs—or the loss of previously present structures. Examples include the reduction or **loss** of wisdom teeth, the **loss** of the palmaris longus tendon in humans, wing **loss** in beetles, vision **loss** in cave fish, **loss** of flight in certain birds, tail **loss** in dogs, feather **loss** in chickens, and the emergence of tuskless elephants. While such changes may confer situational advantages, they represent modifications or reductions of pre-existing features rather than the origination of entirely new ones."

In 2022 science writer Stephen Buranyi writing in the Guardian stated *"A new wave of scientists argues that mainstream evolutionary theory needs an* ***urgent overhaul****. Their opponents have dismissed them as misguided careerists — and the conflict may determine the future of biology. You may recall the gist from school biology lessons. If a creature with poor eyesight happens to produce offspring with slightly better eyesight, thanks to random mutations, then that tiny bit more vision gives them more chance of survival. The longer they survive, the more chance they have to reproduce and pass on the genes that equipped them with slightly better eyesight. Some of their offspring might, in turn, have better eyesight than their parents, making it likelier that they, too, will reproduce. And so on. Generation by generation, over unfathomably long periods of time, tiny advantages add up. Eventually, after a few hundred million years, you have creatures who can see as well as humans, or cats, or owls. This is the basic story of evolution, as recounted in countless textbooks and pop-science bestsellers. The problem, according to a growing number of scientists, is that it is absurdly crude and misleading. For one thing, it starts midway through the story, taking for granted the existence of light-sensitive cells, lenses and irises, without explaining where they came from in the first place. Nor does it adequately explain how such delicate and easily disrupted components meshed together to form a single organ. And it isn't just eyes that the*

traditional theory struggles with. "The first eye, the first wing, the first placenta. How they emerge. Explaining these is the foundational motivation of evolutionary biology," says Armin Moczek, a biologist at Indiana University. "And yet, we still do not have a good answer. ***This classic idea of gradual change, one happy accident at a time, has so far fallen flat.*** *In 2014, eight scientists took up this challenge, publishing an article by Kevin Laland et al 2014 in the leading journal Nature that asked "Does evolutionary theory need a rethink?" Their answer was:* ***"Yes, urgently."*** (Stephen Buranyi 2022)

A central requirement for validating evolutionary theory is the demonstration of observable, testable, and repeatable instances in which genuinely novel anatomical features arise through mutation. However, empirical evidence for such events remains illusive. Most reported cases involve modification, loss, or altered expression of existing structures rather than the origination of entirely new ones. One frequently cited example is the Italian wall lizard (*Podarcis sicula*), introduced to a new habitat, where some populations were reported to develop cecal valves to assist digestion (Herrel et al., 2008). While this case has been interpreted by some as evidence of evolutionary innovation, its status as a truly novel anatomical feature dwindles as new evidence arises. Particularly given the evidence of epigenetic and phenotypic plasticity and the activation of pre-existing developmental pathways.

In the 1970s, researchers transplanted ten adult Italian wall lizards (*Podarcis sicula*) from the small Adriatic islet of Pod Kopište to the nearby islet of Pod Mrčaru (Herrel et al., 2008). Pod Mrčaru is much smaller and originally had far fewer insects but abundant vegetation. Over ~36 years (roughly 30+ generations), the introduced population exhibited striking changes: lizards on Pod Mrčaru shifted to a diet much richer in plant matter (often ~50–60% plants vs. <10% in the source population) (Herrel et al., 2008).

Accompanying this dietary shift were larger head dimensions and much stronger bite forces, advantageous for processing tough leaves (Herrel et al., 2008). Most notably, all dissected Mrčaru lizards — even hatchlings and juveniles — possessed pronounced cecal (ileocaecal) valves in the hindgut, a muscular sphincter and chamber for fermenting plant material (Herrel et al., 2008). In contrast, these valves were absent in the original Kopište population (Herrel et al., 2008).

Mitochondrial DNA confirmed that the two island populations are genetically identical *P. sicula*, indicating that the changes occurred *in situ* rather than via hybridization (Herrel et al., 2008).

In summary, within just a few decades the Mrčaru lizards evolved markedly different head morphology, feeding habits, and gut anatomy (especially the emergence of cecal valves) compared to their source population (Herrel et al., 2008).

- *Key changes (Pod Mrčaru vs. Pod Kopište): increased herbivory; larger, taller heads; higher bite force; longer digestive tract; cecal valves present (in ~62% of individuals) (Herrel et al., 2008; ScienceDaily, 2008).*

Most evolutionary biologists interpret these findings as rapid evolution. Herrel et al. (2008) emphasized that *P. sicula* on Pod Mrčaru had evolved a novel digestive structure within ~30 generations. They reported that "cecal valves, which slow down food passage and provide fermenting chambers," were present in all examined Mrčaru lizards, including hatchlings. Importantly, these valves closely resemble those in other herbivorous lizards (agamids, iguanids, etc.) and had never been seen in *P. sicula* before. Such valves occur in less than 1% of all known lizard species, highlighting their rarity.

Herrel et al. described this as the evolution of a "novel phenotypic character" in this population. They argued that natural selection favored individuals with stronger gut musculature and valves to ferment and digest cellulose from the abundant plants. In their view, the new valves are an adaptive novelty that arose via genetic mutations under the strong dietary shift.

This interpretation was echoed in science communication. For example, National Geographic (2008) reported on the study, calling the new valve a "brand-new structure" that allowed digestion of plant cellulose. The mainstream view holds that the observed gut morphology is an evolutionary innovation: although rare in reptiles, such cecal valves suddenly appeared in *P. sicula* after their introduction. Herrel et al. even noted that their data show "the evolution of novel morphological structures on extremely short time scales." They also found that the valves appeared even in newly hatched lizards, implying a genetic basis for the trait (since juveniles had never themselves eaten plants). In sum, from a secular standpoint this case exemplifies rapid, directional microevolution: within decades, a population gained a previously unseen digestive feature that enhances plant processing.

Other researchers—particularly from a creationist viewpoint—argue that the cecal valves are not truly *de novo* structures but rather an expression of pre-existing genetic potential. They note that many lizards (herbivores) naturally have cecal valves and that *P. sicula* ancestors likely carried the genetic instructions for them. After transplantation, lizards on Pod Mrčaru simply "turned on" these dormant genes in response to a plant-rich environment (Wile, 2010; Menton, 2008). For instance, anatomist David Menton (2008) remarked that the "new" valve is "simply an enlargement of muscles already present in the gut wall," not an entirely new organ. From this viewpoint, the observed valves represent phenotypic plasticity or epigenetic regulation, not novel mutation.

Multiple lines of evidence support the YEC interpretation:

Rapid Reversibility. When Pod Mrčaru lizards were returned to an insect-only diet, their cecal valves disappeared within weeks. Vervust et al. (2010) reported that after just 15 weeks on an arthropod diet, Mrčaru lizards showed total loss of the cecal valves and shortening of the digestive tract. This rapid, reversible change strongly suggests an environmentally induced adjustment rather than a permanent genetic alteration.

1. Prevalence in Herbivorous Populations. Similar patterns occur in other lacertid lizards. For example, Sagonas et al. (2015) found that 62% of island-dwelling Balkan green lizards (Lacerta trilineata) had cecal valves, whereas only 19% of their mainland counterparts did. The island lizards consumed ~30% plants versus ~10% in mainland lizards, indicating that valves correlate with diet. Such data suggest that cecal valves commonly reappear under herbivorous conditions, consistent with a plastic response.

2. Latent Genetic Capacity. Commentators such as Wile (2012) have hypothesized that the cecal-valve genes were already present but methylated ("turned off") in the insectivorous population. Under a new diet, these genes may have become demethylated ("turned on"), expressing the valve anatomy. Methylation-based silencing is an inheritable epigenetic mechanism (Jaenisch & Bird, 2003). In support, Wile notes that the two island populations are genetically indistinguishable based on mitochondrial DNA (Herrel et al., 2008), implying no new information was acquired. From this perspective, natural selection simply favored lizards that by chance had larger gut musculature, allowing the latent valve to manifest, rather than generating novel genetic code.

3. Epigenetic Inheritance. Epigenetic mechanisms such as DNA methylation and histone modifications can stably alter gene expression without changing the DNA sequence (Jaenisch & Bird, 2003). Epigenetic marks can be passed through cell divisions and even to offspring, creating inherited phenotypic changes beyond the base sequence. Thus, inherited methylation patterns could explain how valve-related genes remained "off" in the Kopište lineage but became active in the Mrčaru lizards. Once activated, these traits (like valves) can appear in every generation without requiring new mutations (Wile, 2012).
4. Creationist Analysis. The Answers in Genesis review concluded that, lacking evidence of new DNA sequence changes, this case is "possibly just natural selection acting on pre-existing genetic information" (Menton, 2008; Hendry, 2009; Answers in Genesis, 2008). Both Menton and Hendry speculated that the changes might simply be the lizards' "plastic response to the environment." In this view, the cecal valve is a "backup" organ—an ancestral feature re-expressed under dietary stress, not a product of novel evolution.

5. The Italian wall lizard case has often been highlighted as a textbook example of rapid, mutation-driven evolution of a novel anatomical feature. Yet closer inspection indicates that the observed changes are more consistent with latent genetic potential, phenotypic plasticity, and environmentally triggered epigenetic regulation than with the origin of a truly new structure. The valves' appearance and disappearance with diet changes (Vervust et al., 2010), their occurrence across related lizard species in herbivorous contexts (Sagonas et al., 2015), and expert analysis that they derive from enlargement of existing musculature (Menton, 2008) all support a plastic, regulatory explanation. The cecal valve in *Podarcis sicula* thus exemplifies an adaptive re-expression of a trait already known elsewhere in lizards, rather than a genuine *de novo* innovation.

This raises a broader question. If mutation is indeed sufficient to explain the rise of complex anatomical novelties, one would expect to find not just a rare isolated, ambiguous case, but tens of thousands of clear, well-documented examples of new organs, tissues, anatomy, or systems arising across the animal kingdom over the centuries. Yet despite decades of intensive study in evolutionary biology, such cases remain conspicuously absent (Behe, 2019; Meyer, 2013). This tension becomes evident in discussions of complex organs such as the eye, which are often presented as having independently evolved multiple times across the tree of life (Nilsson, 2009). Yet empirical demonstration of such repeated origins remains limited and contested (Marshall, 2016). The Italian wall lizard valves are frequently presented as strong evidence, but they are easily refuted and are best explained by regulatory flexibility rather than mutation-driven innovation. The lack of abundant, unambiguous examples of novel anatomical features through beneficial mutation underscores a significant gap in the evolutionary paradigm—one that theorists often overlook.

Increasingly, the language of evolutionary biology over the decades has shifted from the expectation of upward, branching innovation to a more modest framework of *"descent with modification,"* emphasizing micro-variation at the terminal tips of phylogenetic trees rather than the Macro-evolutionary generation of novel body plans (Coyne, 2009; Morris, 1974/2012). In effect, this shift concedes that the grand narrative of continual upward branching evolutionary progress, once central in textbooks, does not align with observed biological data.

The *Podarcis sicula* wall lizard case remains a compelling example of rapid organismal change, yet it sits far outside the realm of beneficial mutations producing a genuinely novel anatomical feature that rises to fixation in a population. Secular researchers emphasize novel adaptive morphology—cecal valves as a newly evolved trait—under strong selection (Herrel et al., 2008).

However, substantial evidence—including the valves' reversibility (Vervust et al., 2010), their distribution in related herbivorous species (Sagonas et al., 2015), and built-in plasticity with epigenetic regulation of gut anatomy—highlights pre-existing genetic capacity and adaptability. As one commentary noted, many vertebrates "exhibit considerable phenotypic plasticity in the morphology and physiology of their digestive system," enabling fast switches between carnivorous and herbivorous physiology (Starck, 1999). Thus, while the Pod Mrčaru lizards unquestionably changed to fit a new niche, the mechanisms of change may lie in gene regulation and selection on existing variation rather than the creation of fundamentally new anatomical information (Menton, 2008).

In the Article: **Do we need a new theory of evolution?** By Stephen Buranyi states: "*The case for EES rests on a simple claim: in the past few decades, we have learned many remarkable things about the natural world - and these things should be given space in biology's core theory. One of the most fascinating recent areas of research is known as plasticity, which has shown that some organisms have the potential to adapt more rapidly and more radically than was once thought. Descriptions of plasticity are startling, bringing to mind the kinds of wild transformations you might expect to find in comic books and science fiction movies.*" (Stephen Buranyi 2022)

Jane Braxton Little's article, "Rapid Evolution Changes Species in Real Time," published in Discover Magazine on January 22, 2015, highlights the phenomenon of rapid evolution, where species adapt to environmental pressures at a surprisingly fast pace, sometimes within decades rather than millennia.

- *"Researchers who once assumed evolution required millennia are documenting species adapting in mere decades, or even shorter time frames."*
- *"Adaptation is happening right under our noses, in our lifetimes."*
- *"Species are evolving at speeds that Darwin could not have imagined".*

"I have seen no evidence whatsoever that these [evolutionary] changes can occur through the accumulation of gradual mutations."- Lynn Margulis, as quoted by Charles Mann: Science's Unruly Earth Mother," Science, Vol. 252, 19 April 1991, p. 379.

For much of the twentieth century, epigenetic mechanisms were thought to be confined to "higher" or more complex organisms. Within the framework of deep evolutionary time, prokaryotic life was generally considered too "primitive" to possess such sophisticated regulatory systems. Recent discoveries, however, have disproven this assumption.

In a landmark study, Payne et al. (2018) demonstrated that the thermoacidophilic archaeon *Sulfolobus solfataricus*, which inhabits the hot, sulfur-rich waters of environments such as Yellowstone's Grand Prismatic Spring, possesses robust epigenetic regulation. These organisms are frequently regarded as models of early life, assumed to represent a primitive cellular state. Yet when exposed to progressively acidic conditions, strains of *S. solfataricus* increased their acid resistance nearly 178-fold compared to wild-type populations (Blum, 2019).

Evolutionary predictions suggested that adaptation would result from beneficial genetic mutations in common pathways. Genomic resequencing, however, revealed a striking pattern: the evolved strains carried anywhere from zero to 141 point mutations, but none of these changes were even causally associated with the new acid-resistant phenotype. In one lineage, no mutations were detected at all, despite stable inheritance of the trait (Payne et al., 2018). Instead, the resistant strains displayed what the authors described as heritable transcriptomes whose regulation was inconsistent with mutation.

Strain	Point mutations
SUL120	141
SARC-C	5
SARC-I	0
SARC-O	29

Figure 4 showing SARC-I strain with zero point mutations while SUL120 had 141. Both exhibited the new acidic resistant ability regardless of mutation count.

From an evolutionary standpoint, this result is highly problematic. If *S. solfataricus* truly reflects a primitive organism near the base of life's tree, it should exemplify adaptation through simple mutation and selection—yet it does not. The expectation would be that new, beneficial mutations drive survival under harsh conditions. Instead, the adaptation bypasses mutation entirely, functioning through built-in regulatory mechanisms that suppress mutation and activate latent, heritable changes. This outcome directly contradicts the evolutionary narrative but is entirely consistent with a design perspective, which predicts that even so-called primitive organisms were equipped from the start with robust systems for adaptability.

As Blum, one of the study's senior authors, explained: "*The surprise is that it's in these relatively primitive organisms, which we know to be ancient... We've been thinking about this as something evolutionarily new. But epigenetics is not a newcomer to the planet*" (Blum, 2019). Collectively, the results indicate that the SARC strains acquired heritable transcriptomes whose regulation is inconsistent with mutation, highlighting that adaptability is not a late evolutionary innovation, but an inherent, design-based feature of life.

Importantly, the process involved a reduction in transposition and forward mutation rates, indicating that the direction of change was opposite to the type of genetic novelty predicted by standard evolutionary models (Payne et al., 2018). As Blum, one of the study's senior authors, explained: "*The surprise is that it's in these relatively primitive organisms, which we know to be ancient... We've been thinking about this as something evolutionarily new. But epigenetics is not a newcomer to the planet*" (Blum, 2019).
The findings suggest that the capacity for adaptive, non-mutational inheritance was present even in organisms traditionally viewed as primitive. Rather than arising through gradual mutational processes, the mechanism appears to involve the activation of pre-existing genetic regulation, consistent with an inherent design for adaptability.

> "*Collectively, the results indicate that the SARC strains acquired heritable transcriptomes whose regulation is inconsistent with mutation.*" (Pisco et al., 2018, Proceedings of the National Academy of Sciences)

Laboratory evolution experiments reinforce the conclusion that adaptation rarely arises through the creation of new genetic functions. The long-term *Escherichia coli* evolution experiment has produced dozens of adaptive changes, yet most of the documented beneficial mutations in these bacteria involve the loss or degradation of pre-existing genes rather than the invention of new functions. Recent work with the eukaryotic yeast *Saccharomyces cerevisiae* paints a similar picture. **Chen and Zhang (2020)** designed an experiment to test how environmental changes affect molecular evolution.

Starting from a single haploid progenitor, they evolved 12 replicate populations under ten different constant conditions and another 72 populations in changing environments that rotated among either five antagonistic or five concordant conditions.

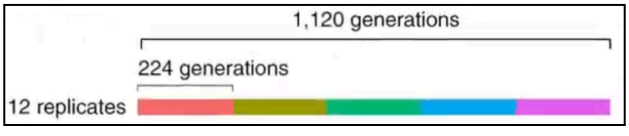


Image 9.

Each population was propagated for 1,120 generations, with frozen samples taken every 56 generations. They then sequenced the genomes of all final populations and compared the ratio of nonsynonymous to synonymous single-nucleotide variants (SNVs), denoted ω. In antagonistic changing environments—where a mutation that is beneficial in one condition is likely deleterious in another—ω was significantly lower than in the corresponding constant environments. The reduced ω arose because beneficial nonsynonymous mutations that increased in frequency during one environmental phase were purged when the environment changed, a phenomenon known as antagonistic pleiotropy. The authors observed similar patterns when they examined nonsense mutations and frame-shifting insertions/deletions, which are often sources of advantageous mutations in experimental evolution. These findings demonstrate beneficial mutations in natural settings because they fail to fix when conditions fluctuate. They also reveal that many of the advantageous changes involve gene disruption, not the creation of new gene functions.

Study details and outcome

- Growth in the presence of the carcinogenic dye Congo Red
- Exposure to copper ions
- An alkaline environment (pH 8)
- Oxidative stress from hydrogen peroxide
- Treatment with the strong antibiotic neomycin

To test adaptation in fluctuating environments, replicate cultures were cycled successively through these five conditions, each for 224 generations, for a total of 1,120 generations.

By the end, the populations that experienced all five conditions had far fewer *net* genetic changes than expected from simply adding together the mutations that appeared during each phase. In other words, many mutations that rose in one environment were lost in the next. This confirmed the principle of antagonistic pleiotropy: mutations that are advantageous in one setting may be disadvantageous in another, and so they rarely persist when the environment shifts.

Chen and Zhang concluded that beneficial mutations are often undercounted in fluctuating environments because they emerge temporarily but fail to fix in the population. Crucially, the mutations that *did* help survival in these conditions were

overwhelmingly gene-disrupting rather than gene-building. In other words, adaptation here arose by degrading or silencing pre-existing functions.

This result dovetails with many earlier laboratory evolution studies, including the long-term *E. coli* experiments, which likewise showed that adaptation most often proceeds through loss or inactivation of genes. The fact that the pattern is seen in both prokaryotes (*E. coli*) and eukaryotes (yeast) suggests a general rule: under selective pressure, organisms frequently adapt by breaking or down-regulating existing functions rather than inventing new ones.

Because destructive mutations occur far more quickly than constructive ones—estimates suggest 100 to 1,000 times faster—these "quick-fix" degradative mutations dominate the early stages of adaptation. Over time, they can also crowd out slower, potentially constructive changes, locking populations into a path of functional reduction.

The study design intentionally rotated environments every 224 generations to prevent any mutation from reaching full fixation, highlighting how unstable beneficial changes can be when conditions fluctuate. But had the time between switches been longer, many gene-loss mutations would have become permanent, limiting the yeast's future adaptability.

Taken together, these findings underscore a key insight: adaptive mutations in experimental evolution almost always come from loss or reduction of function. Constructive, novel functions appear—if at all—only on vastly longer timescales, while destructive changes sweep populations within months. This universal tendency toward devolution presents a serious challenge to evolutionary expectations of innovation by random mutation and natural selection.

FOUNDER EFFECT

These cases represent only a preliminary example of the limitations inherent in evolutionary explanations. The following section examines predictions derived from the founder effect, demonstrating once again how empirical evidence fails to align with evolutionary expectations. **Frank Lewis Marsh (1899–1992)**, a Seventh-day Adventist biologist and young-earth creationist, developed the concept of the *baramin* (created "kinds") in his books *Fundamental Biology* (1941) and *Evolution or Special Creation?* (1947). Marsh's framework implied that only a small number of certain "kinds" were preserved and survived—consistent with a genetic bottleneck scenario derived from the biblical account of Noah's flood.

In his later book *Evolution, Creation, and Science* (1944), Marsh elaborated that interbreeding (hybridization producing viable offspring) is a key indicator that organisms belong to the same baramin. He emphasized *discontinuity systematics*—the idea that there are fixed reproductive and morphological boundaries between kinds, and that variation happens within, not across, these boundaries. Modern baraminology continues this line of thought, with proponents arguing that the founder effect, high rates of hybridization within kinds, and observed genetic discontinuities confirm Marsh's predictions (Paley, 2002).

Even after the discovery of the founder effect, evolutionists posit that populations cannot bottleneck down to a single pair and recover since small populations are highly vulnerable to random loss of alleles and beneficial traits can vanish purely by chance. So with only two founders, there's almost no buffer against drift — so the gene pool shrinks even faster. So evolutionary minded scientists publish studies suggesting a minimum viable population for mammals is in the hundreds to thousands to sustain long-term survival. The Bible tells us otherwise, that 2 of every unclean animal went onto the ark. Therefore it is a prediction of ours that evolutionary population genetics are wrong and species have the ability to recover as long as the bottleneck is a single generation, there is rapid expansion and population growth. This biblical founder effect has already proven to be true time and time again from different animal, reptile and aquatic life. For example, just a single pair of Mouflon sheep (ovis aries) were left on the most isolated locations in the world: the Kerguelen Sub-Antarctic archipelago. After 46 years since the introduction, the population reached 700 and heterozygosity increased. They noted that this "exceeds the range predicted by neutral genetic models and stochastic simulations." The title of the study even admits their shock and surprise; **Unexpected heterozygosity in an island mouflon population founded by a single pair of individuals (Renaud Kaeuffer et al 2006).** As you can see, "unexpected" is right in their title. This is but 1 of many examples.

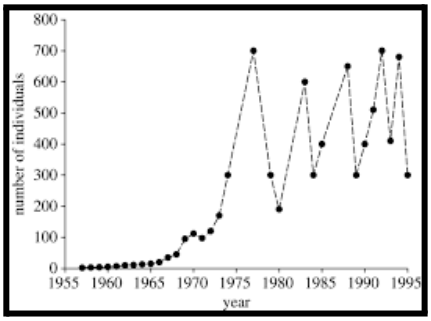


Figure 5. Number of individuals estimated on the Kerguelen mouflon population.

We see this same thing in insects (BBC, 2016), birds (Grant & Grant, 2002), deer (YLE, 2018), rabbits (CNN, 2022), fish (National Geographic, 2008), bison (National Geographic, 2020), wolves (missing source), whales (missing source), lizards (Herrel et al., 2008), and yes even humans (Soodyall et al., 2003; Macgregor et al., 2009).

Difference pedigree vs Phylogeny

In 1962 (Zuckerkandl & Pauling): Proposed the first idea of a molecular clock — mutations accumulate at a roughly constant rate, reinforcing tree-like branching. This concept led to early predictions based on fossils.

Evolutionists assume that many humans split or diverged from a chimp-human ancestor around 6 - 7 million years ago from the fossil record. Based on this, they calibrated a mutation rate clock to match this idea. This rate is known as the phylogenetic mutation rate.

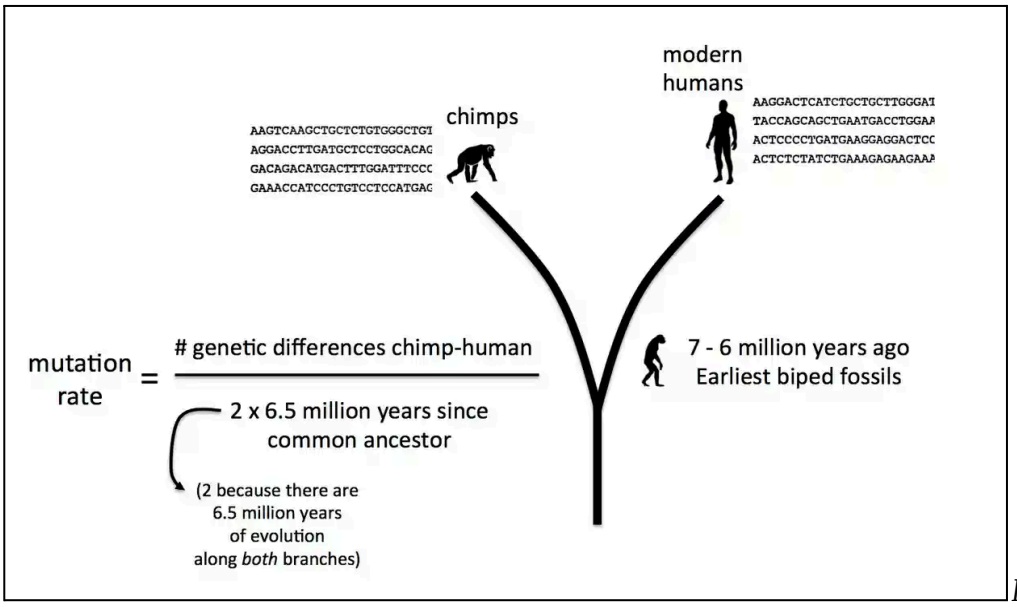


Figure 1. Phylogenetic mutation rate calculation based on assumed ancestor split 6 - 7 million years ago.

"In a typical study, divergence between homologous genomic regions across a phylogeny is ***calibrated with fossils*** *or other external criteria at specific nodes in order to determine substitution rates"* (Thorne, et al. 1998; Sanderson 2002; Thorne and Kishino 2002; Drummond, et al. 2006; Yang 2006), relying on the **assumption** that the rate at which substitutions accumulate between species is equal to the mutation rate **for neutral sites** (Kimura 1983). Thus, in principle, phylogenetically-based and pedigree-based methods **should** produce equivalent estimates of the mutation rate. The reality is however, they do not.

Biblical creationists believe that mankind and animal life was created around 6,000 - 10,000 years ago. A heavy contrast to the evolutionary timeframe. Therefore we would predict a rapid mutation rate that would place Eve, the mother of all living - within that timeframe. To do this, we do not want just a mutation rate but a substitution rate.

- **Mutation rate** = how often new mutations appear in individual genomes per generation.
- **Substitution rate** = how often those mutations actually become **fixed in the population**.
- **Homoplasmy** = occurs when all mitochondrial DNA (mtDNA) molecules within a cell or organism share the same nucleotide at a specific position. This state can arise when a mutation becomes fixed, either by genetic drift or selection, replacing the previous variant entirely.

Using the evolutionary rate of change *(based on a primate split divergence)*, *"it is predicted that ~1/590 children will differ from their mother at some site within this span -or, in other terms, that the rate of mutation in this span of the mitochondrial coding region is 1 mutation/590 generations."* 1/590 is for that specific coding-region span (per-locus) equivalent to roughly 1 substitution arising every 14,750 years, given a per-generation substitution rate of =0.001694 (Howell et al 1996 - Page 505). The reality is, the exact opposite was discovered by Howell.

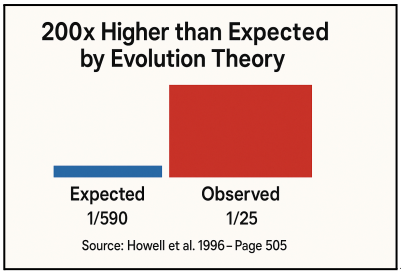
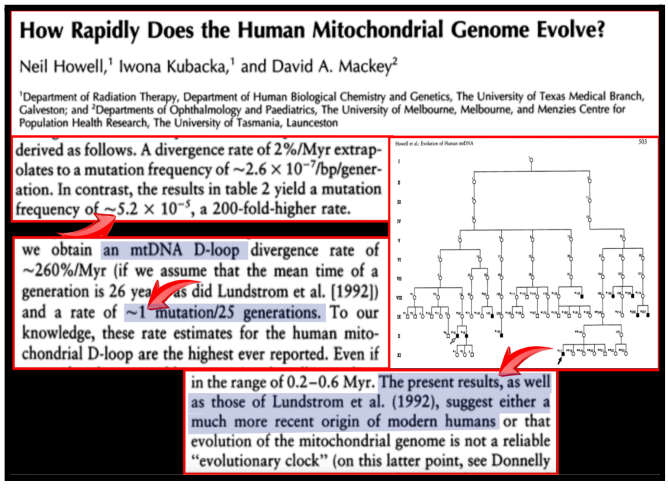


Figure 2. Observed vs Evolutionary Estimated rates.

Observed Mutation Rate


How Rapidly Does the Human Mitochondrial Genome Evolve?

Neil Howell,[1] Iwona Kubacka,[1] and David A. Mackey[2]

[1]Department of Radiation Therapy, Department of Human Biological Chemistry and Genetics, The University of Texas Medical Branch, Galveston; and [2]Departments of Ophthalmology and Paediatrics, The University of Melbourne, Melbourne, and Menzies Centre for Population Health Research, The University of Tasmania, Launceston

derived as follows. A divergence rate of 2%/Myr extrapolates to a mutation frequency of ~2.6×10^{-7}/bp/generation. In contrast, the results in table 2 yield a mutation frequency of ~5.2×10^{-5}, a 200-fold-higher rate.

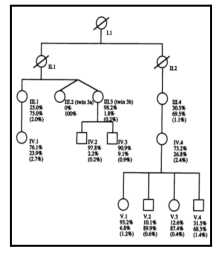
we obtain an mtDNA D-loop divergence rate of ~260%/Myr (if we assume that the mean time of a generation is 26 years, as did Lundstrom et al. [1992]) and a rate of ~1 mutation/25 generations. To our knowledge, these rate estimates for the human mitochondrial D-loop are the highest ever reported. Even if

in the range of 0.2–0.6 Myr. The present results, as well as those of Lundstrom et al. (1992), suggest either a much more recent origin of modern humans or that evolution of the mitochondrial genome is not a reliable "evolutionary clock" (on this latter point, see Donnelly

The first observable pedigree-based mutation rate study carried out by Howell et al (1996) found *"the results in table 2 yield a mutation frequency of ~5.2 x 10-S, a 200-fold-higher rate."* Equating to 1 per 25 generations (1/25) gen = 0.0424 per 1.2-kb D-loop locus per generation *(≈3.53×10^{-5} per site per gen if divided by 1,200 bp)."* This mutation rate was far faster than evolutionary estimates derived from phylogenetic divergence based on the fossil record, confirming the earlier suspicions of Lundstrom 1992. The participants were ethnically diverse

European and Australian. *"In the first analyses, the entire 1.2-kb D-loop sequence was determined for 36 members of the TAS2 pedigree and for four members of the three English 14484 LHON pedigrees who have the TAS2 haplotype. For these 40 individuals, we analyzed DNA that had been isolated from venous blood samples or from dried blood spots."... "The TAS2 Australian LHON family spans 12 generations and comprises > 700 maternally related individuals whose genealogy can be unambiguously traced back to a woman born in 1714 in London. In the first analyses, the entire 1.2-kb D-loop sequence was determined for 36 members of the TAS2 pedigree (fig. 1) and for four members of the three English 14484 LHON pedigrees who have the TAS2 haplotype. For these 40 individuals, we analyzed DNA that had been isolated from venous blood samples or from dried blood spots."* This substitution rate going back hundreds of years in pedigree generations confirmed early suspicions (Lundstrom 1992) of a rapid mutation rate.

Then in 1996 K.E. Bendall et al published their findings; *"we calculated that the rate of mutation and fixation in the first hypervariable segment of the human mtDNA control region is between 1.2 x 10^{-6} and 2.7 x 10^{-5} per site per generation. This range is in good agreement with published estimates calculated by other methods."* This study is a 4 - 5 generation deep pedigree and resulted in a substitution rate of 4/360 or 1/90 for HVR1 only, meaning across both hyper variable regions they would've found twice the amount of mutations resulting in 1/45. As you can see, the results confirmed the rate is extremely fast.



Soon after in the following year, Steven Mumm and his team tested the 410 base pairs (bp) in the D-loop region. They traced two pedigrees back **5 generations' deep** and obtained a rate of 0.0410 mutations per generation or 1 x 10^{-4} . For HVR1 this is 1/59 with a divergence rate of 1.51. Extrapolating his results again to both hyper variable regions again, this works out to be another perfect example of a rapid substitution rate at 1 substitution every 30 generations or 1/30.

The same year Thomas Parsons, a forensic scientist with access to the FBI's large blood bank database, conducted a massive pedigree study from diverse people groups that confirmed similarly high mitochondrial substitution rates across broader human samples.

Thomas Parsons and his team conducted the largest pedigree-based substitution rate study to date analyzing mitochondrial DNA from blood samples of diverse people worldwide. *"90 African-American (53, FBI: 40, AFDIL), 115 Afro-Caribbean (ESS), 114 Sierra Leone African (provided by C. Ginther), 90 Hispanic (provided by C. Ginther), 100 British Caucasian (FSS), 233 European American (AFDIL). Our database represents random, unrelated individuals."*

They concluded:

> *"Our observation of the substitution rate, 2.5/site/Myr, is roughly* ***20-fold higher than would be predicted from phylogenetic analyses.****"* (Parsons et al., 1997)

The authors further noted that if this empirical pedigree rate were used to calculate the mitochondrial DNA molecular clock, it would place the most recent common ancestor (MRCA) of human mtDNA at only ~6,500 years ago—a figure far younger than standard evolutionary timescales and directly with the Biblical timeline.

Parsons stated: *"We compared DNA sequences of two CR* (control region) *hypervariable segments* (HVR1 & HVR2) *from close maternal relatives, from 134 independent mtDNA lineages spanning 327 generational events. Ten substitutions were observed, resulting in an empirical rate of 1/33 generations, or 2.5/site/Myr."* This result was obtained by observing 10 substitution differences after 327 generational links.

Since this study was wide scope, the results sent shockwaves through the evolutionary community. Many researchers initially denied it, others attempted to explain it away, and still others directly refused to believe it. The FBI adopted the results regardless of drama or criticism.

Figure 3. FBI Forensic Science Application using Parsons et al 1997 mutation rate.

The following year, Parsons and his colleagues were joined by fellow geneticist critic Holland who repeated the study using different samples. Altogether, 10 of leading geneticists took part: David S. Muniec, Kevin Sullivan, Nicola Woodyatt, Rosemary Alliston-Greiner, Mark R. Wilson, Dianna L. Berry, Koren A. Holland, Victor W. Weedn, Peter Gill, and Mitchell M. Holland. They did rigorous testing, checking each hyper variable region 1,000 times each! They confirmed the earlier rate published in 1997 in two blood samples, one from the *"National institute of Health and Mental Health of mixed ancestry"* and also samples from the *"Forensic Science Centre"*.

Table 1 • Point mutations between positions 1–370 of the mtDNA CR

	No. of families	Sample type	Country of origin	Comments	Point mut. observed*	Total no. of meiosis
Parsons *et al.*	73	blood	USA	Samples from Natl. Inst. of Health and Mental Health Mixed ancestry	4	121
Parsons *et al.*	5	blood	England	Samples from Forensic Science Centre	1	32

Figure 4. Taken from Parsons et al 1998 study showing results from both samples.

The team reported and published a mutation rate of **1 substitution every 30 generations**. This implied a mitochondrial DNA MRCA for all humans of only 6,000 years ago, dramatically younger than the standard evolutionary timescale and lands on the popular young earth creation date for Eve and the secular evolutionary community exploded.

As science journalist Ann Gibbons (1998) noted in Science, in an article titled Calibrating the Mitochondrial Clock:

- *"But solving the mystery of the Romanov's remains raised another puzzle that **first troubled forensics experts and is now worrying evolutionists**."*

- *"It could also **complicate the lives of evolutionary scientists** who use the mtDNA mutation rate as a clock to date such key events as when human ancestors spread around the globe."*

- *"**Evolutionists have assumed** that the clock is constant, ticking off mutations every 6000 to 12,000 years or so."*

- *"Regardless of the cause, **evolutionists are most concerned about the effect of a faster mutation rate.** For example, researchers have calculated that 'mitochondrial Eve' the woman whose mtDNA was ancestral to that in all living people - lived 100,000 to 200,000 years ago in Africa. Using the New Clock, she would be A MERE 6,000 YEARS OLD."*

1: These major substitution rate studies trying to obtain the MRCA for humanity look inside the D-Loop *(Control Region)* of the mtDNA, **not** the coding region because purifying selection and strong background selection would render it a horrible clock over time.

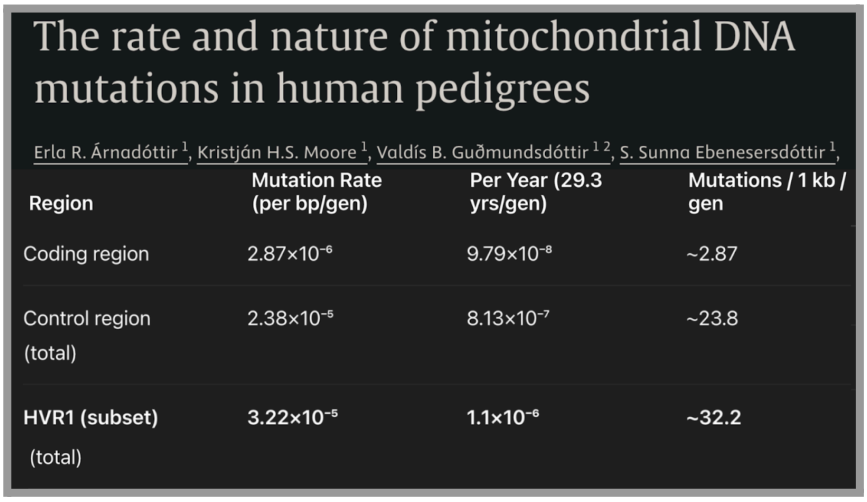
Instead, geneticists look at the best areas in the mtDNA with the weakest selection to obtain the most accurate clock. These regions are called hypervariable regions, specifically HVR1, HVR2.

Ka-Kei Sam 2021 quotes: "*The mitochondrial control region has a higher evolutionary rate and tends to* ***be under weaker purifying selection than protein-coding genes****, allowing the region to accumulate length variations more readily (Tang et al., 2006; Resch et al., 2007).*"

Just look for yourself on the following page all the studies that specifically focus on this region. As you can see, the experts realize these regions are superior and make the best clocks and this is why most studies focus on them.

PEDIGREE DATA SETS	REGION ANALYZE
Lundstrom et al 1992	D-Loop (Control region)
C M Koehler et al 1991	D-Loop (Control region)
Howell et al 1996	D-Loop (Control region)
Bendall et al 1996	HVR1
Mumm et al 1997	HVR1
Soodyall et al 1997	D-Loop (Control region)
Parsons et al 1997	D-Loop (Control region) HVR1 + HVR2
Parsons et al 1998	D-Loop (Control region) HVR1 + HVR2
Cavelier et al 2000	HVR1 + HVR2
Sigurdardottir et al 2000	D-Loop (Control region) HVR1 + HVR2
Bowling et al. 2000	D-Loop (Control region) HVR1
Savolainen et al., 2000	D-Loop (Control region)
Heyer et al 2001	HVR1 + HVR2
Lambert, D.M. et al. 2002	D-Loop (Control region) HVR1
A T Bowling et al 2002	D-Loop (Control region)
Cristina Luis et al 2002	D-Loop (Control region)
Howell et al 2003	D-Loop (Control region) Pooled studies
Pamela A Burger et al 2004	D-Loop (Control region)
Klütsch et al 2010	D-Loop (Control region) HVR1
Madrigal et al 2012	HVR1
Kurushima et al. 2012	D-Loop (Control region) HVR1 & HVR2
Hudson et al. 2017	D-Loop (Control region)
Shedko 2017	D-Loop (Control region)
Dell et al. 2020	D-Loop (Control region)
Andy C Dell et al 2020	D-Loop (Control region)
Menéndez et al 2023	D-Loop (Control region)
Agnar Helgason 2024	Entire mtDNA, Coding, Control (D-loop), HVR1

Agnar Helgason et al. (2024). Nature Communications confirmed this as well by showing that the hypervariable region vs control region vs the coding region in mtDNA all mutate at different rates. Helgason's team published the different mutation rates as seen below in *figure 5* below based on a deep rooted pedigree of 119,211 people going back as far as 1510 or 17 generations ago. The rates are different based on the power of selection between the two compartments *(Coding/Control)*. The coding region is highly conserved, while the entire D-loop (control region) is not. The Hypervariable regions inside the D-Loop follows this neutral trend as well.



The rate and nature of mitochondrial DNA mutations in human pedigrees

Erla R. Árnadóttir [1], Kristján H.S. Moore [1], Valdís B. Guðmundsdóttir [1 2], S. Sunna Ebenesersdóttir [1],

Region	Mutation Rate (per bp/gen)	Per Year (29.3 yrs/gen)	Mutations / 1 kb / gen
Coding region	2.87×10^{-6}	9.79×10^{-8}	~2.87
Control region (total)	2.38×10^{-5}	8.13×10^{-7}	~23.8
HVR1 (subset) (total)	**3.22×10^{-5}**	**1.1×10^{-6}**	**~32.2**

Figure 5. From 2024 Helgason et al confirmed that the control region (D-loop) is **near neutral** and the HVR is completely neutral with no conserved bp regions. The control region mutates approximately **8.3 times faster** than the **coding region** and **10x faster than HVR1.**

So based on these rates and the fact that Heyer et al found only 40 sites total in the D-loop *(1,122 bp)* mutate (3.6%) and these hotspots within the D-Loop *(HVR1 & HVR2)* are functionally neutral where selection has no effect in this region making them the best internal clock regions and highlights why the HVR regions in the D-Loop remains the gold standard for studying mitochondrial variation, ancestry and origins. Why? Because the control region doesn't code for proteins, only a few of these regions even perform replication or transcription within it and they make up only a small portion of the entire sequence, contrary to the coding region which is why the D-Loop region is far superior than entire mtDNA for a clock in studies. Since selection is inadequate in most of the mtDNA D-loop the rate of change is fast and mutation hot spots experience essentially no selective constraint since purifying selection is not occurring, validating them the best possible clock in the genome. Tests have shown that the famous hot spots *(e.g., 16093, 16129, 16311, 16519)* experience almost no purifying selection. The only selection we find occurring in the D-loop region is a small set of base pairs, 151 in total and none of them are mutation hot spots. HVRs contain no conserved regions at all, the region is entirely neutral. Making it even better than the D-loop which is already a good clock.

Recap

- HVR hotspots mutate a lot because they are not under strong functional constraints, so changes rarely affect anything essential making the entire region the best for testing.

- Only 18 base pairs in HVR1 and 22 bp in HVR2 show variation hot spots and the mutations that occur are benign and persist over generations. The overall D-loop

mutates a little slower because there are overall more base pairs with less regions mutating (functionally constrained).

- Lethal or highly harmful mutations never show up in population samples because those embryos would not survive, so we don't count them in pedigree studies anyway.
- Hot spot mutations persist across generations, validating they are not harmful and even low level purifying selection is negligible in the D-loop and Hypervariable regions.
- Therefore overall mutations in these regions are considered neutral by geneticists, and are not seen by or removed by purifying selection which is why they persist over generations and make the best "clock" for ancestry.

2: Even diverse **deep rooted pedigree** studies confirm Parsons' published MRCA date. They have shown that no matter how deep the pedigree is, the results and rates stays the same. From single generation dyad studies to multi-generational pedigrees, the rate is always the same. This tells us the D-Loop & hypervariable regions are functionally neutral with no selection or background selection taking place. Therefore invoking selection of any kind to slow this clock down, just tells us that critics of these rapid observed rates do not even realize what region of the mtDNA is being looked at in these studies, since all these studies listed here focus specifically on these regions, just for that reason.

Our next study is Brandstätter et al. (2004) by the International Journal of Legal Medicine. The goal of the study was to assess the mutation rate in the entire human mtDNA control region *(the full D-loop: HVR-I + HVR-II + central region ≈ 1,122 bp)* in maternal pedigrees relevant to forensic genetics. Brandstätter and colleagues conducted their pedigree analysis primarily on European individuals, specifically Central European Caucasians. Their samples came from: Austria, Germany, and Switzerland). What they found: Each mother–child comparison represents one transmission event. They tested 135 transmissions in total and found 3 substitutions, 135 transmissions = 0.0222 mutations per transmission. This is expressed as = 1 substitution per 45 generations *(since 1 / 0.0222 = 45)*. They also sometimes normalize this to 1.6×10^{-6} mutations/site/generation for the 1,122 bp region. Their rate therefore reflects heteroplasmic mutation appearance, not fixation, so similar to Parsons *(1997/1998),* but slightly lower because of course they tested the entire D-Loop not just HVR1 and HVR2 segments. Since those 3 substitutions were specifically found in HVR1 & HVR2, when corrected for the smaller 676 bp regions actually sequenced, the observed rate of 1 substitution per 45 generations becomes roughly 1 substitution every 27 generations (1/27). Right in line with Howell and Parsons.

The study Santos et al. 2005 Titled: *Understanding differences between phylogenetic and pedigree-derived mtDNA mutation rate: a model using families from the Azores Islands (Portugal). They looked at 321 maternal transmissions, covering 973 bp of the D-loop (which includes both HVR1 + HVR2), and detected 11 substitutions in 321 transmissions. That gives a raw familial rate around 1 mutation every 29 generations, the same as Parsons (1997/1998).* With a pedigree depth average of 5.8 generations, Santos explicitly explored what happens when you compare the raw data vs. filter and exclude certain mutational variants. They showed that: "*Depending on how transitions are classified (germline vs. somatic/artifactual), the rate can vary by almost an order of magnitude.*" Santos only was able to slow down the mutation rate by filtering out what they thought may have been somatic and heteroplasmy, but in reality were removing actual mutations. They admit their own shortcoming and misclassifications: *"It is not possible to distinguish with certainty between mutations arising in the germ line and those arising post-zygotically, since a mutation that occurred early in embryonic development* ***could be transmitted through the germ line of the next generation****."* And: "*Some* ***mutations classified here as somatic could in principle be germline events that were not detected in the mother because of low heteroplasmic levels below our detection threshold.****"* They go on to note that the detection limit in their sequencing protocol was high, meaning any germline variant present at lower frequency could easily have gone unseen as well. Since heteroplasmies can turn into fixed substitutions, then observing and counting them and accounting for such a thing is important. This is why when we see studies like Connell 2022 who set a lower detection rate to detect them after a few generations classified them as true mutations. Low and behold, they got the same rate as Parsons yet again. Santos further cautions that: *"Our approach is therefore conservative;* ***the true germline mutation rate may be somewhat higher than the corrected estimate****."* Their *"corrected rate"* is thus a **lower bound**, not a definitive rate. While their **raw observed rate** became their upper bound. The reason they favored conservative reporting was to align short-term pedigree estimates with long-term phylogenetic expectations, as stated in the paper. So once again it's the paradigm *(evolutionary mindset)* driving the conclusions. They explicitly stated that **their filtering strategy intentionally erred on the side of undercounting**, explaining that the true germline mutation rate it's probably much higher towards the raw data rate, but they preferred a lower, more evolutionarily friendly slower rate. Again, they admitted that their rate after filtering is most likely off, and the observed rate aligns perfectly yet again with Parsons and the others. In summary, Santos et al. removed or reclassified certain substitutions simply to adjust observed pedigree rates to phylogenetic evolutionary timescales.

Ann Gibbons First International Workshop on Human Mitochondrial DNA, 25 to 28 October 1997, Reports: *"By tracing the mutations back through the family pedigree, Howell was able to estimate that both mutations probably arose in the same woman who was born in 1861, yielding an overall divergence rate of one mutation every 25 to 40 generations."* ***Both of our studies came to a remarkably similar conclusion,"*** says Howell, whose study

was published in late 1996 in the American Journal of Human Genetics. Both also warned that phylogenetic studies have *"substantially underestimated the rate of mtDNA divergence."* **The fact that the raw substitution rate of Santos et al landed directly on Parsons, Howells, Brandstätter and Lundstrom's rate speaks volumes.**

Side by side comparisons: Regions tested: Parsons (Entire HVR1 & HVR2). Santos 2005 (Scanned the entire D-loop which included HVR1, HVR2 & HVR3, but removed substitutions assumed somatic and ignored hotspots in HVR1 & HVR2. Since these hotspots are really the only true regions that mutate while the other 95% does not, it is obvious now why after filtering they went from the observed average rate *(1/29)* to a slower rate. The team literally observed that certain base substitutions appeared in parallel in unrelated maternal lines, a classic hallmark of recurrent hotspot mutation, which they *"filtered out"* on purpose to align with evolutionary phylogenetic assumptions. Not to forget that they had a detection level limit of 10% and anything below that was ignored or removed.

Our next study is Madrigal et al., 2012 Titled *"High mitochondrial mutation rates estimated **from deep-rooting** Costa Rican pedigrees." in the* American Journal of Physical Anthropology. Focused on multiple large families colonial-era Costa Rican and Colombian pedigrees of mixed ancestry mixed of European + Amerindian ancestry and they explicitly filtered the data: they computed rates both "unfiltered" *(all mutations)* and "filtered" *(removing known hotspot sites and suspect lineages)*: The research used several large and detailed family trees (19 in total), rather than a single family unit. In terms of generations, that represents on the order of about **9–12 generations deep or 270–360 years** average tracing back to colonial founders in the 17th century, in pedigree terms this is really deep, though in animals we can do better. They report Observed substitutions / transmissions (a*fter excluding clear adoptions*): 7 / 273 Per-generation substitution frequency (HVR-I): 7/273 = 0.02564 per locus per generation; Mean generation time used: 28.3 years. Per-site rate (per Myr): 0.02564 gen^{-1} ÷ 360 bp = 7.12×10^{-5}/site/gen ÷ 28.3 yr/gen = 2.51×10^{-6}/site/yr or 2.51 per site per Myr *(In the paper they call that "divergence rate"* (2×μ). Their final divergence rate (2.51×10^{-6}) or 2.51/site/Myr, which is effectively the same as Parsons' 2.5/site/Myr. The raw per-generation frequencies differ slightly (0.0256 vs 0.0306) because *(a)* Parsons assayed both HVR-I+II (676 bp) while the Costa Rica study counted only HVR-I (360 bp), and *(b)* different generation times (20 vs 28.3 yrs) are used in the per-site conversion. Once you normalize to per-site per Myr, they match. The bare minimum slowest rate they could obtain **through filtering** was 2/220 per-locus per generation because they removed clear regions where mutations tick fast such as Hotspot sites and they also removed in Pedigree 3 because it had an an ambiguous site *(could be a mutation or adoption)*, they also removed Pedigree 11 mutation *(16182)* again a known hotspot as well.

So after removing multiple HVR1 hot spots they obtained a slow minimum mutation rate, while the unfiltered mutation rate again lands directly on Parsons, Howells, Lundstrom, Bendall, Mumm, Heyer, Helgasons, Santos and many others.

	Point estimate	0.995 lower	0.95 lower	0.95 upper	0.995 upper
Max mutation frequency (7/273) [a]	0.0256	0.0084	0.0127	0.0519	0.0652
Min mutation frequency (2/220) [b]	0.0091	0.0012	0.0028	0.0323	0.0450
Max mutation rate [c]	2.51×10^{-6}	0.71×10^{-6}	1.08×10^{-6}	4.40×10^{-6}	5.52×10^{-6}
Min mutation rate [c]	0.89×10^{-6}	0.12×10^{-6}	0.27×10^{-6}	3.17×10^{-6}	4.42×10^{-6}

Figure 6. [a]Top: Max observed substitutions 7 out of 273 excluding pedigrees 2 and 17. Total meioses 289 − 11 − 5 = 273. [b]Minimum rate **after filtering** by excluding pedigrees 2, 3, 11 and 17, and the mutation at site 16,335. Total meiosis 289 − 11 − 43 − 10 − 5 = 220, generation time = 28.3 years, **MRCA 6,226 years ago**.

Once again, the data converge on the same numbers, year after year, decade after decade, study after study. The odds of such precise agreement arising by chance are statistically improbable. At this point, it's no longer coincidence, it's a pattern carved into the very fabric of the data itself. The region is as near neutral as can be obtained and the only way these studies give altered results are if we look at single shallow pedigrees, few samples, high filtering, calibrating and adjusting the numbers via phylogenetic inference, or outright ignoring observed mutations.

Since all we have to do as creationists is empirically show how the mutation rate can explain the data from our Biblical timeline, which we can do and evolution cannot. Then it is fair to say that our model is superior at explaining the diversity we see. We can also explain the diversity of all life on earth as well because of these rates & we make predictions on it. Only by adjusting the data through evolutionary assumptions can they ever even remotely get any older evolutionary dates. If they took away evolutionary assumptions or did not calibrate their clocks by anchoring them to historical dates, they would never have any evidence at all. Meaning, without force fitting and retrofitting the data through circular reasoning - evolution has no evidence at all in the realm of mutation rates. This shows the sad and pathetic affair evolution is in, but the public is unaware of all of this because anytime you were to search online for any of this, it is only ever going to give you the phylogenetic evolutionary rates as though they are a fact when in reality, the exact opposite is true.

Next, Heyer et al 2001. This pedigree study depth is great, with 16 different pedigrees ranging mostly around 9 - 12 generations deep. The study resulted in: *"For the HVI sequences, we obtained* (MRCA) *220 generations or 6,600 years, and for the HVII sequences 275 generations or 8.250 years."* Combining HVR1 & HVR2 we get 7,425

years, or 247 generations as the midpoint. This rate is basically the same as Parsons, but since Parsons used a 20 year generation time and Heyer used a 30 year generation time, the dates are a little different. Adjusting it to Parsons for this study would be; 247 generation x 20 yr/gen = 4,940 years ago. As you can see, the rate is similar to Parsons if not faster even though the diverse pedigrees go back many generations, refuting critics who say the rate slows down over time from selection. Though Heyer published a slower rate of 0.0079 per generation per 673 bp, *(95% CI 0.0023–0.186)* they admit in the paper this is because they excluded the oligo-C tract around 308–315 in HV2 which had an additional 5 visible mutations. They also did not count any transient heteroplasmy, but only mutations that rose to near-homoplasmy in at least one sampled individual were scored. These are major reasons why they published such a slow rate.

Our final study is the Árnadóttir & Helgason et al. (2024) study. The dataset contained 2,059 mother-to-child transmissions in its largest matriline, the actual *vertical* depth of that pedigree was 17 generations *(Icelandic ancestor born in 1520)*. This study came more than twenty-five years after Parsons's original 1997 discovery, yet the results fall right next to each other when expressed on the same scale. This new deCODE pedigree analysis *(64,806 individuals in 116,663 transmissions)* published and reported: "*Our control region estimate is around* ***8 times higher than for the coding region****, at* $\mathbf{2.38 \times 10^{-5}}$ *mut/bp/gen (95% CI 2.30 –* $\boldsymbol{2.47 \times 10^{-5}}$*)* or 8.13×10^{-7} mut/bp/year *(95% CI 7.85–8.43 ×* 10^{-7}*)* and ***falls within the range of previous pedigree-based estimates***, $5.17 \times 10^{-8} – 2.63 \times 10^{-6}$ mut/bp/year. This is consistent with the Parsons et al. (1997) pedigree estimate once his reported divergence rate *(*2.51×10^{-6} */site/year)* is converted to a true mutation rate ($\mu \approx 1.26 \times 10^{-6}$ /site/year). Remember, Árnadóttir et al. used a 29.3 year maternal generation time, while Parsons used the traditional 20-year generation typical of 1990s pedigree work. That difference alone changed Helgason's MRCA age by roughly one-third. They also imposed a 5% read-frequency threshold, meaning any heteroplasmic variant below that level was invisible and ignored in their analysis. Those two methodological choices, longer generations and stricter detection criteria, when accounted for narrow the apparent gap even further. When translated into a more intuitive form, Parsons observed roughly one substitution every 33 generations on average, whereas Helgason et al. observed about one every 42.5 generations on average. However, using their observed fastest confidence interval *(CI)* ***the rate is 1/36 or 0.0277 subs/gen*** across the same hypervariable regions, essentially within the same order of magnitude after accounting for thresholds and generation-time adjustments. Helgason also conveniently published the HVR1 mutation rate as well: 3.22×10^{-5} per/bp/gen or 1.1×10^{-6} per year 29.3 generation time. Considering the vast leap in sequencing technology and sample size (*from 327 transmissions in 1997 to over 116,000 today),* the enduring similarity between these rates underscores how robust Parsons's early pedigree signal really was. Using the upper confidence bound of the

Árnadóttir & Helgason et al. (2024) control-region rate (2.47×10^{-5} mut/bp/gen, over ≈1,122 bp), we see an effective substitution rate of 1 per 36 generations, right next to Parsons's classic 1 per 33 generations 1997 published average. Under identical assumptions about control-region divergence, Parsons's 1/33 rate yields an MRCA of 6,500 years, whereas the high Árnadóttir–Helgason et al rate lands right next to Parsons at 7,090 years ago, demonstrating how closely the two independently derived rates converge even though studies are decades apart and technology has improved drastically. It is clear and obvious that the D-Loop is mutating at a neutral rate and the clock is true over time.

3: The average substitution rate published by Parsons was not the fastest rate he obtained. So critics who say that you can only get a 6,500 year age for Eve if you use the fastest rate or only look at single generation pedigrees are either ignorant of the study or lying. Here is the breakdown of each pedigree depth Parsons team catalogued.

AFDIL; Average pedigree depth 2 generations.

Oxford; Average pedigree depth 6.4 generations, multi-generation pedigrees (***Published results***) 1/32.8 = 1/33

CEPH; Average pedigree depth 5.9 generations.

Amish; Average pedigree depth 2 generations.

Parsons' study resulted in two independent dyad pedigrees *(single generation mother to daughter)* resulting in both a fast and slow rate (AFDIL & Amish). Parsons and his team included both published results using the mean average, matching the Oxford results which came from the deepest pedigree to guarantee the most accurate substitution rate. If Parsons had published only his **fastest rate observed (1/17).** Mitochondrial Eve would have lived as recently as **3,400 years ago**. The **published rate** by Parsons *(1/33 in 1997 & 1/30 in 1998)* is around what we see time and time again across multiple pedigrees decade after decade from studies looking at the same regions regardless of pedigree depth since purifying selection and background selection is insufficient there. This is what makes this evidence so strong in favor of the Biblical timeline and why critics invoking selection or maybe they were accidentally counting somatic mutations are not valid rescue devices. So even if Parsons was a bad study overall, it is just one out of many we can look at to get a big picture and that is exactly what we have done here in this study. One thing remains true, regardless of pedigree depth in the D-Loop, results return the same.

Pedigree depth of studies: Howell 1996 = 11 generations, K.E. Bendall 1996 = 5 generations, Mumm 1997 - 5 generations, Parsons 1997 *(Oxford Pedigree)* published rate = 6 generations, Parsons 1998 (2-7) Brandstätter 2004 = 4 generations, Madrigal et al. 2012 = 9–12 generations, Heyer et al 2001 = 32 generations, Santos 2005 = 5.8 generations, Connell et al. 2022 = 4 generations, Árnadóttir & Helgason 2024 = 17 generations.

4: Just when you thought it could not get worse for evolution, we have to talk about when a "multiple hit" happens. This occurs when:

- A site mutates once…
- and then later mutates again (to another base or back to the original base).

Result: This would cause the observed mutation rate to appear slower than the true mutation rate because you can't see the earlier mutation in that same place anymore.

- You cannot count all the mutations that actually occurred.
- Observed differences between lineages stop increasing linearly (Diversity stops increasing).
- The molecular clock slows as time increases.

Remember, the clock in this region is already way too fast for evolution, and now there is another underlying factor that possibly makes the rate even faster if accounted for. Unfortunately this factor largely goes unnoticed and therefore unaccounted for, but it favors the Biblical timeline even more.

Literally the best clock in the genome, which is neutral and selection free *(the hypervariable regions inside the D-loop),* lands on the Biblical timeline time and time again and that's using the **average** rate of change based on pedigree studies of all depth. Not just in humans either, but **all living species ever tested as well**.

Large, ethnically diverse pedigree studies, **regardless of depth**, worldwide have all obtained rates that fall within a timescale consistent with the Biblical chronology, not the deep-time evolutionary model. Only one framework can be true, and the empirical pedigree data cannot be ignored or excused away. Indeed, the FBI has long used these pedigree based mutation rates in forensic casework to identify victims and convict criminals going deep into the past. If the rates were fundamentally wrong, such life-and-death applications would collapse, but they have proven robust and reliable over decades. Parsons et al. *(1997 & 1998)* remains the most ethnically diverse pedigree-based mtDNA rate studies ever performed. It combined 134 lineages from multiple global ancestries via the U.S. Armed Forces DNA Identification Laboratory (1997) to ensure population-wide relevance and again the following year *(1998)*.

When critics cannot refute the results, they desperately quote people like Parsons' or Ann Gibbons' who we will show you below what they state.

Parsons: *"Using our empirical rate to calibrate the mtDNA molecular clock would result in an age of the mtDNA MRCA of only ~ 6,500 ya.,* ***clearly incompatible with the known age of modern humans****.*

Based on what? Their evolutionary mindset views the past, that is all. Let's keep reading.

*"Even acknowledging that the MRCA of mtDNA may be younger than the MRCA of modern humans[35], **it remains implausible to explain the known geographic distribution of mtDNA sequence variation by human migration that occurred only in the last ~6,500 years**."*

Notice that they had to admit that there is no way to explain how the MRCA could have lived 6,500 years ago based on "known" *(assumed based on radiometric dating actually)* geographic distribution of humans and migration around the world during this time according to their conventional population and migration views of the past. They even said that applying their pedigree rate *"would yield an unrealistically young age."* Yes, unrealistic in evolutionary terms, but from a young-earth perspective, this data is not unexpected. If humanity was created between 6,000 and 10,000 years ago, such as that described in Genesis, the resulting patterns of mitochondrial diversity could have formed within a much shorter timescale than evolutionary models allow. What mainstream researchers obtained in these pedigree studies reflect a historically recent human history and falsify evidence of deep evolutionary ancestry and this is what confuses secular scientists to this day. Different labs, different families, different centuries, yet the numbers keep matching. The probability of that being random? Astronomical. Yet, the critics of the data will choose that over the alternative.

Ann Gibbons reports regarding the 6,000 year date: *"No one thinks that's the case,..."* Oh, you mean the secular evolutionary community that all think alike? What a surprise. No wonder she wrote an entire article talking about how desperate evolutionists were regarding these new observed rates landing on the Biblical timeline.

Ignoring the published results and running to the commentary shows me that the critics are desperate to hand wave away these results and would rather believe someone's personal opinion regarding their views of the past rather than what the empirical evidence and conclusions show. This is desperation at its finest and the exact opposite of good science, yet common in the evolutionary community. They are willing to throw away the observed empirical evidence all because it does not conform to their belief system that they want to be true. The funny part about that is, when the critics started to attack Parsons over the data and treating him like a creationist, he challenged them all back. He dared them to refute his work or to even come to his lab themselves, which is exactly what Holland did in 1998, all to literally confirm his work from the year prior which they accepted as valid. We are left with only internet atheists saying these rates are wrong now, which is the red flag everyone should notice.

Selection

Purifying selection is just a rescue device used by critics who do not know much about the subject talking to an audience who know even less. The reality is, selection has nothing to do with the difference between these observed pedigree study results and

the phylogenetic evolutionary rates. Why? Because as we have discussed, all these pedigree studies I mention focus on the HVRs or the entire control region which is under extremely weak selection at only 151 bp sites. Howell et al (2003) study quotes; *"Sigurdardottir et al. (2000) conclude that* ***selection is unlikely to be a major factor that underlies the difference between phylogenetic and pedigree divergence rates****...", "At this point, the results suggest that* ***selection has not preferentially distorted the pedigree mutation spectrum relative to the phylogenetic one****."*

Howell et al. (2007) — "For most sites in the control region, the relative rate of substitution was similar to the rate of neutral evolution (assumed to be most closely approximated by the substitution rate at 4-fold degenerate sites)." This suggests most mutations in the control region are probably harmless and **neutral**, not affected much at all by selection.

Sigurðardóttir 2000 *"Only if there is selection acting directly on the sites ... can population-genetics effects be invoked as a cause of systematic differences between pedigree and phylogenetic estimates."* They considered this, but rejected the idea that selection bias explains the differences. They also note that "*except at sites on which selection is acting directly, phylogenetic and pedigree approaches are estimating the same quantity*." That is a clear statement that selection is only acting directly at specific sites and not a pervasive background-selection (BGS) effect across the whole control region.

Background selection is the process by which purifying selection reduces genetic diversity at **linked sites**. Purifying selection is the removal of deleterious *(harmful)* mutations from a population, while background selection is the resulting reduction in genetic diversity in neutral regions of the genome that are physically linked to the sites under purifying selection *(Ivana Cvijović 2018)*. So think of it like this; a new harmful mtDNA mutation arises, it gets seen and removed by purifying selection because it reduces fitness. While that mutation is being removed, all neutral variants near it (linked sites) also disappear because mtDNA is non-recombining. This is Background selection. So if purifying selection rarely occurs at all, then background selection cannot work and thus has no effect at all. Since deleterious mutations rarely arise in the D-Loop, selection is extremely rare and weak. In HVR's it is non-existent. Therefore invoking background selection to slow the rate down when deleterious mutations are either rarely ever arising in the region or not at all, tells me the critics know very little about this topic. If purifying selection is not removing deleterious mutations, then background selection cannot operate, because background selection is the population-level consequence of purifying selection acting on linked sites.

No purifying → nothing to remove → no background selection effect → no linked neutral variants get removed.

Strong purifying selection occurs because deleterious alleles arise often and are quickly removed, this causes strong background selection to also be strong like we find in the coding region.

With weak purifying selection - deleterious alleles linger, you have weak background selection. Since the mutations arising in the D-loop are functionally neutral, then background selection will be having no effect at all. These features make the region mostly neutral or nearly neutral because most mutations in non-coding sequences have no strong effect on fitness. For the 3.6% of the base pairs mutating that are functionally neutral, there is no purifying selection occurring, meaning there is no background selection occurring either. For the rest of the entire 1,122 bp D-Loop, only 151 bp total are conserved and these regions rarely mutate at all.

For neutral sites *(such as the d-loop & HVRs)* the substitution rate equals the mutation rate. This was Kimura's prediction and conclusion and it still holds true today. Since we are dealing with the neutral hypervariable regions, then invoking selection to slow the rate down is irrational. Neutral substitution rates only depend on generations, not on population size or growth affect variance. The *neutral* substitution rate *(the rate at which neutral mutations become fixed)* is determined primarily by the neutral mutation rate (μ), not by population size or time. In a strictly neutral model, k = μ, meaning the substitution rate equals the neutral mutation rate *(independent of population size)*, because small populations fix mutations faster by drift but generate fewer new mutations overall, and large populations do the opposite. Those effects cancel out. Population size and gene flow can change which sites behave neutrally or how quickly alleles move between populations, but they don't change the overall neutral substitution rate itself. The true neutral substitution rate per generation stays constant and equals the neutral mutation rate *(μ)*. Time by itself doesn't *cause* the rate to change either, so adding time each generation still has the same chance of fixing a neutral mutation. Therefore selection cannot slow down a neutral region since it cannot act upon it, so adding more time does nothing to help slow the rate down in these regions. The fact is as stated earlier HVR1 & HVR2 the most common sites used because of their small neutral regions and mutational hot spots that have extremely weak purifying selection rarely occurring in only a total of 40 bp sites for both HVR's that mutate. This becomes the best clock to use in the genome for ancestry and when tested and shows the timeline is Biblically based and far from any evolutionary timeline. Therefore when we look at the coding region, rates are erratic and all over the place. Completely inaccurate across the board as seen published in different studies. This is why the D-Loop being nearly entirely neutral is a way better clock and is ticking 8.2x faster. As stated briefly either, only a few regions inside it are conserved, the RS regions (94 bp) and the CSB regions *(CSB I, II, III = 57 bp)* cluster which stands for Conserved Sequence Blocks are small regions *(151 bp total)* that undergo selection. While these regions rarely ever mutate, to get the purest clock we

go to the HVR's, they contain the smallest subset of base pairs that mutate and all mutations arising are purely neutral, which no bp compartment conserved at all, making them by far the best clock in the genome. For these HVR regions, the substitution rate equals the mutation rate 100% which the D-loop is not far behind. Studies looking at the entire mtDNA or the coding region cannot make this claim. Selection is seen to remove most mutations in the coding region, especially early on after they arise. The longer they persist the more of a chance they have to remain, but many get weeded out and removed early on either from selection or drift.

Multiple papers (e.g., Soares 2009; Santos 2008; Howell 2003, Helgason 2024) all show:

- Much higher substitution rate in the D-loop than the coding region.
- Very low selective constraint.
- No detectable background selection beyond a few conserved motifs (mentioned above).

This matches the expectation of geneticists who focus on the control region to obtain an accurate historical clock. Whatever selection exists in mtDNA, none of it is coming from the hypervariable regions - confined in multiple published papers. For more details on this subject including our own mutation rate study, see our paper; **When Facts Falsify Fossils: Pedigree Substitution Rates Upend the Evolutionary Timeline.**

The Y-chromosome mutation rate provides further evidence consistent with a young-earth creation (YEC) framework. Jeanson (2017), working with Answers in Genesis, predicted from the biblical timeline of Noah's flood that pedigree-based mutation rates should converge on a timeframe of approximately 4,500–5,300 years ago. Early pedigree studies did not support this prediction, as they reported relatively slow mutation rates with only 12.4–15.5 sequence coverage (Xu et al., 2009; Helgason et al., 2015). However, Karmin et al. (2015) conducted the first high-coverage sequence analysis of the Y chromosome and revealed that earlier studies had missed thousands of mutations, thus significantly altering the estimated mutation rate.

Karmin et al. (2015) did not emphasize the faster Y-chromosome mutation rates in the main body of their publication, but instead reported them in the supplemental data rather than the body of the paper. These high-coverage results indicated rates approximately ten times faster than phylogenetic evolutionary estimates, consistent with patterns also observed in mtDNA pedigree studies and orders of magnetite faster than evolutionary phylogenetic rates. In response, the authors noted the need to *"explore additional filters,"* effectively favoring data that aligned with the established evolutionary timeline while sidelining results that suggested significantly faster mutation rates.

A recent bottleneck of Y chromosome diversity coincides with a global change in culture - Supplemental Text

March 2015

Project: genographic

S2). However, the application of the regional filters led only to a modest reduction of false positive calls judged by the number of father-son/brother-brother (FS) differences and the count of recurrent mutations (Table S2). The number of FS differences was approximately 10 fold higher than the expected number of de novo mutations considering the range of published Chr Y mutation rates (Xue et al. 2009; Francalacci et al. 2013; Mendez et al. 2013; Poznik et al. 2013). This finding prompted us to explore additional filters.

Image 11.

Maretty et al. (2017) essentially confirmed Karmin's earlier findings by employing the same high-coverage sequencing methods. Unlike low-coverage approaches, which frequently miss mutations, high-coverage scanning repeatedly surveys the Y chromosome, thereby uncovering more mutations with each pass. Using this method, results consistently show approximately two to three mutations passed on per generation. When coalescent calculations are performed—taking into account the maximum differences observed today (about 440 mutations)—the results yield only 146 generations. With a 30-year generation time, this places the most recent common ancestor (MRCA) at approximately 4,380 years ago, while a 35-year generation time extends the estimate to about 5,110 years ago. These findings are incompatible with the standard evolutionary timeline but align closely with young-earth creation (YEC) predictions.

Additional lines of evidence have been proposed to corroborate these results. Jeanson (2020) applied his Y-chromosome molecular clock to Amerindian population history, suggesting a major migration into the Americas around 800 AD, with potential links to eastward expansions from the Roman world into China. More recently, Jeanson (2022) expanded on this work in *Traced: Human DNA's Big Surprise*, integrating Native American historical sources such as the Red Record—containing genealogies, migration accounts, and time units based on sachem reigns—alongside archaeological, linguistic, and haplogroup data to support a recent origin and diversification of human Y-chromosome lineages.

Image 12

By tracing genealogies recorded in the Red Record and counting the reigns of Indian chiefs documented as "sachems", Jeanson (2022) calculated a timeline consistent with a crossing

of the Bering Strait region into the Seward Peninsula, Alaska, around 900 AD. Using a mutation rate of three per generation, this estimate closely aligns with his earlier prediction that Native Americans migrated into the region around 800 AD (Jeanson, 2020). In the historical sciences—including archaeology, genetics, and geology—dating methods rarely achieve precise single-year accuracy, often carrying margins of error spanning decades or even centuries. Thus, a result within a century of the predicted value supports rather than falsifies the model.

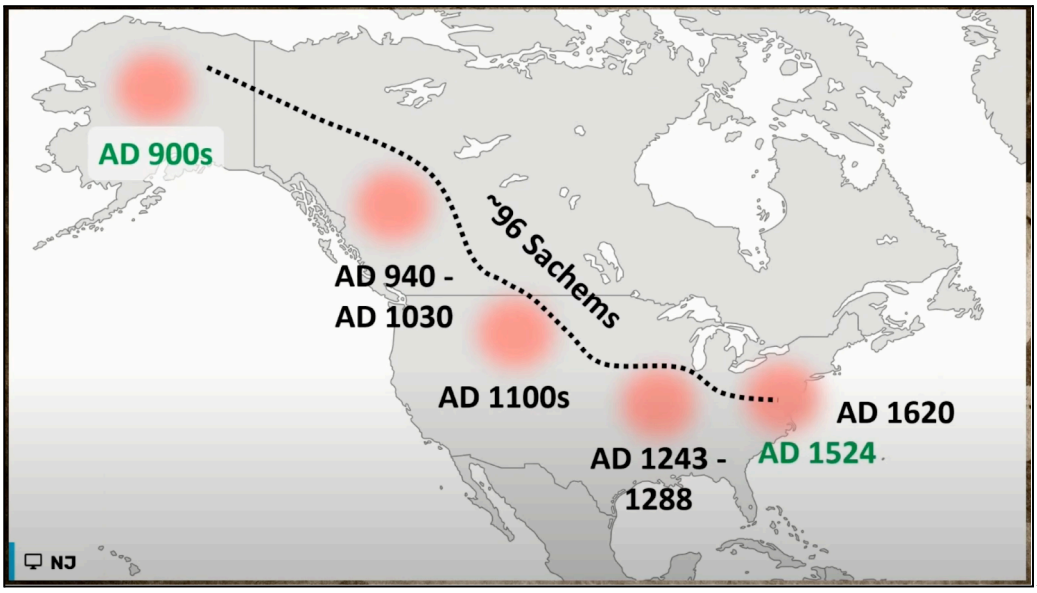


Image 13. Migration map of native Americans documenting their travels through Sachems (the time a chief ruled) documented in the Red Record.

Had the prediction been entirely incorrect—such as if the migration had taken place thousands of years earlier as evolution poses—the model would not be supported and the prediction failed. Instead, the proximity of the calculated result to the prediction demonstrates explanatory power within the expected range of historical uncertainty.

Another notable aspect of the Y-chromosome mutation rate concerns its correlation with biblical genealogies. When applying the observed rate of approximately three mutations per generation, the cumulative total from Noah's lineage corresponds to the generation of Peleg. In the biblical record, Peleg's lifetime is associated with the division and expansion of human populations from the Middle East (Genesis 10:25). The alignment of genetic calculations with this specific generational marker has been interpreted by young-earth creation researchers as further support for the concordance between pedigree-based mutation rates and the biblical timeline (Jeanson, 2017, 2022).

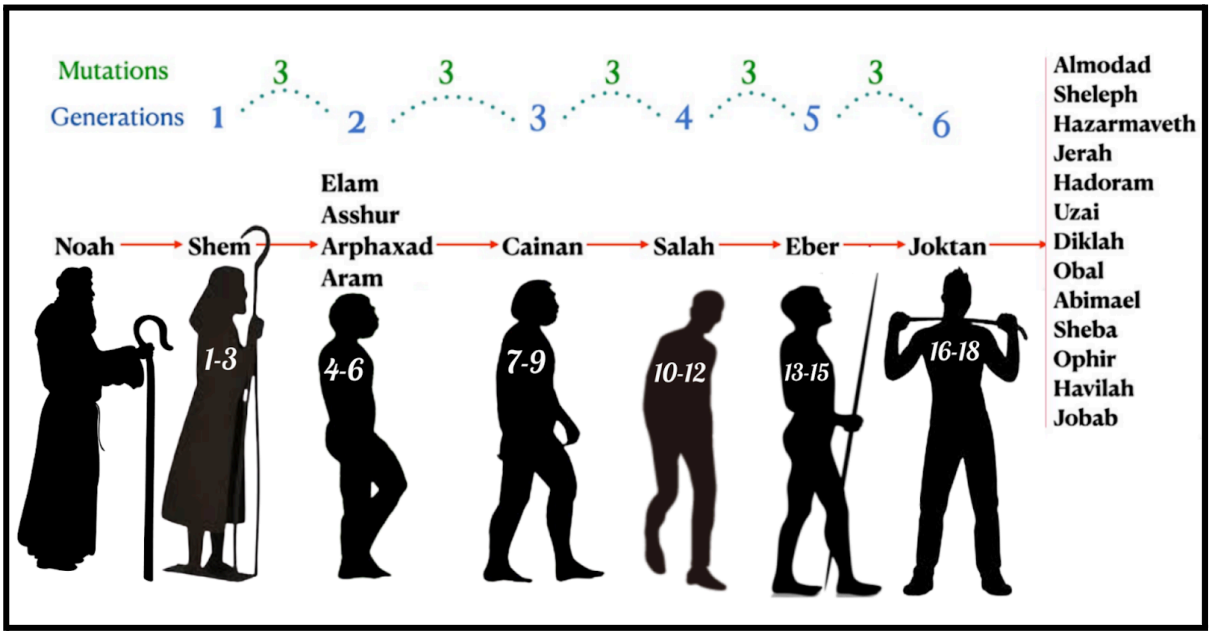


Image 14.

Further evidence for a recent Y-chromosome MRCA comes from the observation that haplogroups appear to arise in the MRCA who had approximately 16 mutations prior to the onset of branching. This pattern suggests that the founding lineage carried a small set of initial mutations before subsequent diversification occurred. Such a result is consistent with expectations from a single common ancestor within the biblical timeframe and supports the interpretation that haplogroup branching reflects post-Flood population expansion rather than deep evolutionary history (Jeanson, 2017, 2022).

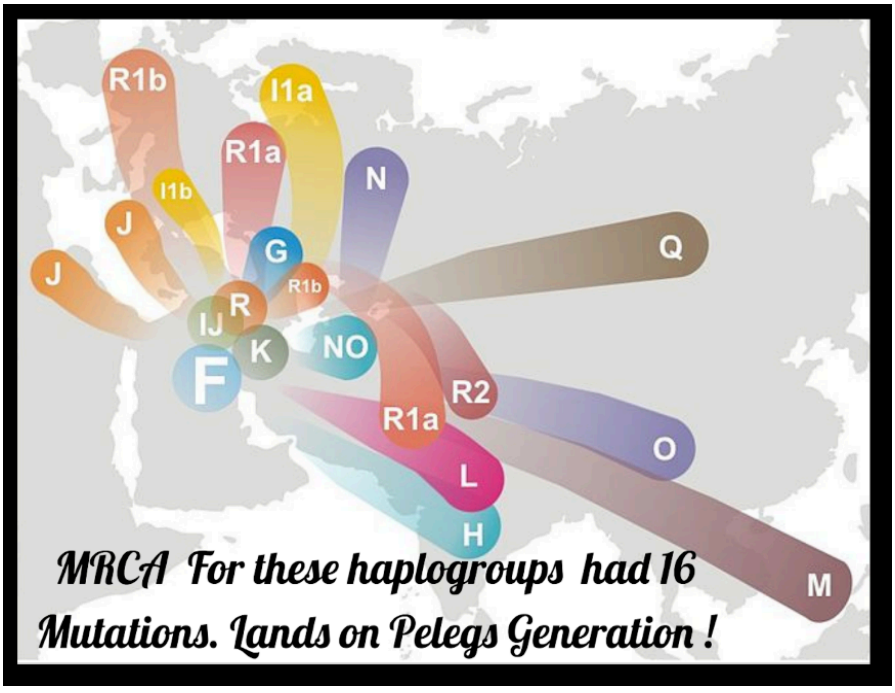


Image 15.

This timing of expansion is further supported by large-scale genomic analyses. Tennessen et al. (2012), in their study Evolution and Functional Impact of Rare Coding Variation from Deep Sequencing of Human Exomes, reported that the maximum-likelihood estimate for accelerated human population growth occurred approximately 5,115 years ago. This finding places the onset of rapid expansion within the same timeframe as Y-chromosome coalescent calculations, providing additional evidence for a recent, post-Flood growth of the global human population.

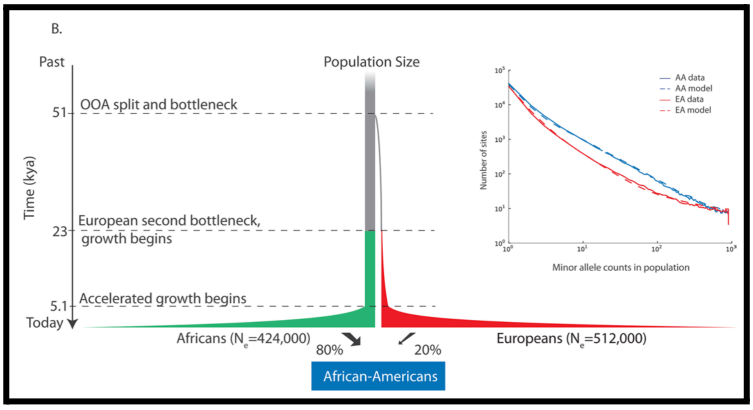


Figure 11 from the 2012 study showing the number of individuals tested and the number of alleles over time based on population size and when population growth began.

These mutation-rate estimates align not only with migration patterns and the formation of haplogroups, but also with empirically derived human population growth rates. Tallavaara et al. (2021) reported that simulated annual population growth rates ranged between 0.4% and 3%, values more consistent with ethnographic data than with traditional archaeological estimates. Even at the lowest end of this range (0.4%), the growth trajectory is sufficient to account for the current global population beginning from eight individuals approximately 5,100 ± 300 years ago (Nailor M. 2025). This convergence of genetic, demographic, and historical evidence further strengthens the argument that such findings are unlikely to be the result of mere statistical coincidence.

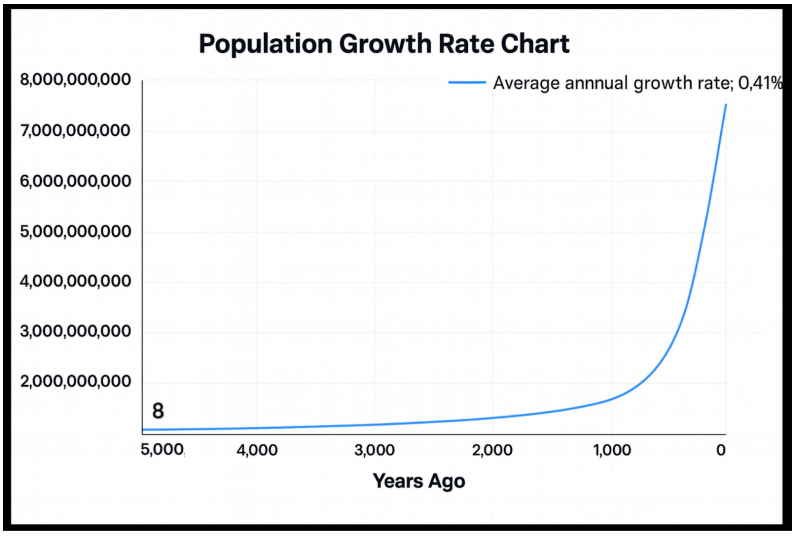


Figure 12. 0.4% population growth rate

Genealogical simulations further corroborate these findings. Rohde, Olson, and Chang (2004), in their study *Modelling the Recent Common Ancestry of All Living Humans,* used both a probabilistic model and computational Monte Carlo simulations to examine patterns of ancestry across human populations. *Their results suggest that the genealogies of all present-day humans overlap extensively in the recent past, with the most recent common ancestor (MRCA) of all living humans appearing just a few thousand years ago. Interestingly, their models consistently placed this MRCA in eastern Asia. These independent analyses, while not explicitly framed in a biblical context, nevertheless converge with predictions of a recent MRCA as expected within a young-earth creation (YEC) framework.*

You can even find other new study titled: Post-Flood Populations: Haplogroup formation and Fixation Dynamics in from Noah to Babel Dispersion by Nailor, M. 2025 where I run a simulation replaying the tower of Babel story from scripture to show how all of the genetic evidence fall right into place.

Mutation Saturation

What other confirmations do we have that can affirm and solidify the YEC model even more? Additional support comes from mutation saturation predictions. Jeanson (2015) reasoned that if humans had diverged from a primate ancestor with chimpanzees seven million years ago, the accumulation of mitochondrial DNA (mtDNA) mutations should have long since reached saturation—meaning new mutations would simply overwrite existing ones in the same nucleotide positions, obscuring further diversity. Based on expected rates, approximately 21,457 mutations should have arisen, with saturation occurring at around 12,000 mutations. Yet empirical data show only 1,483 mutations, far below saturation. This disparity supports Jeanson's argument that mtDNA diversity has accumulated over only a few thousand years, consistent with a recent origin of humanity as described in Scripture.

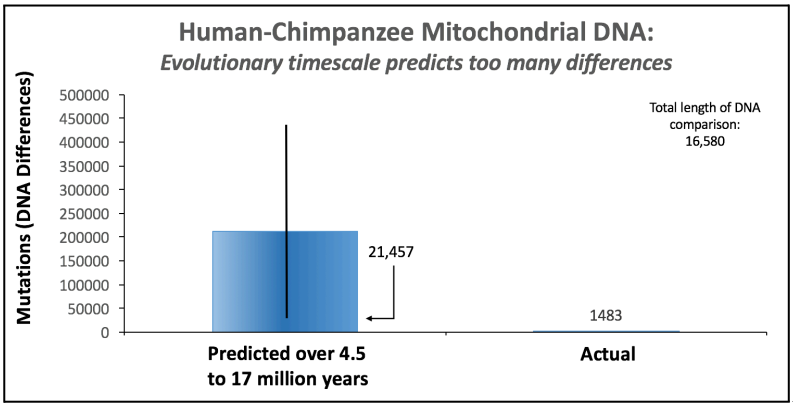


Figure 13. Mutation saturation chart.

This same lack of mutation saturation is observed across the animal kingdom, providing further evidence that places a young-earth timestamp on the origin of life. Taken together, these results raise a fundamental problem for evolutionary theory: if its foundation rests on mutation and selection, yet it cannot reliably model or predict

mutation rates, the framework is undermined. Indeed, evolutionary theorists themselves have acknowledged the inability to make accurate predictions of mutation rates within their paradigm.

By contrast, real-world applications rely on pedigree-based mutation rates rather than evolutionary phylogenetic estimates. For example, the National Institutes of Health (NIH) employs Mendel's Accountant to simulate and track the accumulation of mutations, including those related to cancer, across populations (National Cancer Institute, 2020).

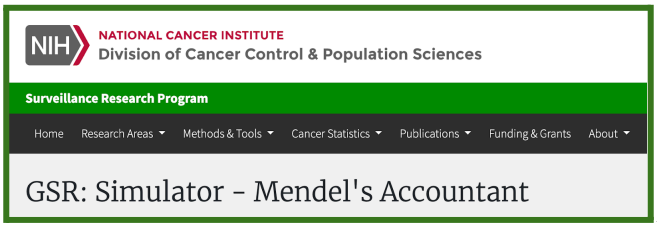


Image 16

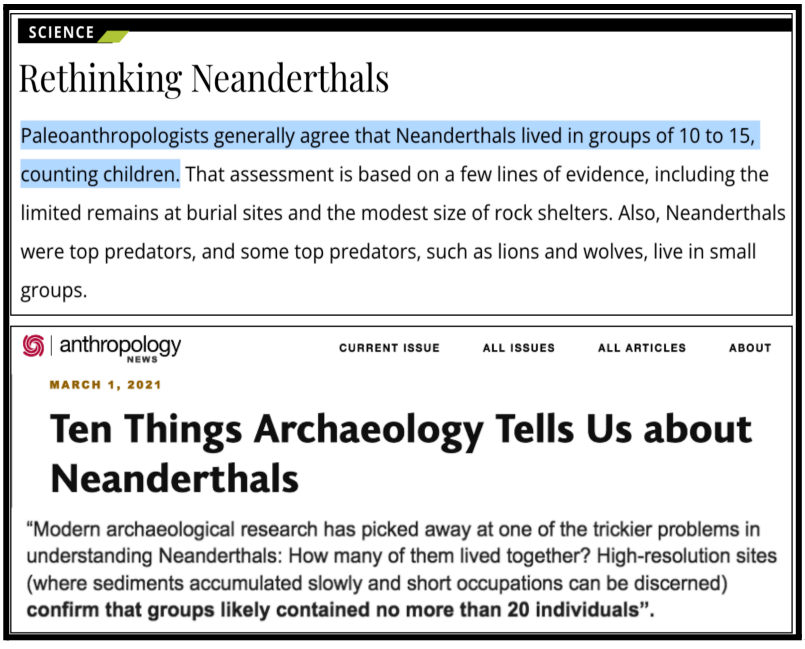
Similarly, the Federal Bureau of Investigation (FBI) utilizes pedigree mutation rates in forensic genetic analysis, not evolutionary phylogenetic rates. These institutional choices underscore the practical validity of pedigree-based mutation models over evolutionary ones.

Image 17.

Taken together, this body of evidence underscores the fragile state of evolutionary theory, which, critics argue, would have been discarded long ago were it not institutionally protected. Across the living world, genetic diversity remains consistently low, with few fixed mutations despite high observed mutation rates. In humans, only 24 total substitutions have become fixed across the entire population. This pattern—rapid substitution rates coupled with a small number of fixed substitutions—is irreconcilable with deep-time evolutionary expectations. Instead, it points toward a recent global bottleneck and a recent creation date. Simulation studies confirm this interpretation, demonstrating how haplogroup formation and fixation dynamics align with a post-Flood dispersal model (Nailor, M. 2025).

Is there additional evidence we can look at regarding supposed early hominins like Neanderthal, Denisovan, Heidelbergensis and Erectus not agreeing with the evolutionary timeline? Yes, a study was recently published by PLOS biology titled: **Inbreeding, Allee effects and stochasticity might be sufficient to account for Neanderthal extinction**. It states: *Our results indicate that the disappearance of Neanderthals might have resided in the smallness of their population(s) alone: even if they had been identical to modern humans in their cognitive, social and cultural traits, and even in the absence of inter-specific competition, Neanderthals faced a considerable risk of extinction.* Their numbers show something unpredicted and a falsification of deep time.

The findings indicate that small population sizes would have driven groups such as Neanderthals to extinction within only 500 years, with an upper limit of about 10,000 years. This conclusion is based on well-established principles of population size and inbreeding, both of which characterized Neanderthal populations. As reported in *Science* magazine's feature "Rethinking Neanderthals" and in the *Anthropology News* article "Ten Things Archaeology Tells Us About Neanderthals," Neanderthals typically lived in groups of only 10 to 15 individuals, including children, with an upper bound of no more than 20 at a time (Gibbons, 2008; Smith, 2018). These small, isolated, and inbred groups align with population dynamics that predict rapid decline and eventual extinction.


SCIENCE

Rethinking Neanderthals

Paleoanthropologists generally agree that Neanderthals lived in groups of 10 to 15, counting children. That assessment is based on a few lines of evidence, including the limited remains at burial sites and the modest size of rock shelters. Also, Neanderthals were top predators, and some top predators, such as lions and wolves, live in small groups.

anthropology NEWS

CURRENT ISSUE ALL ISSUES ALL ARTICLES ABOUT

MARCH 1, 2021

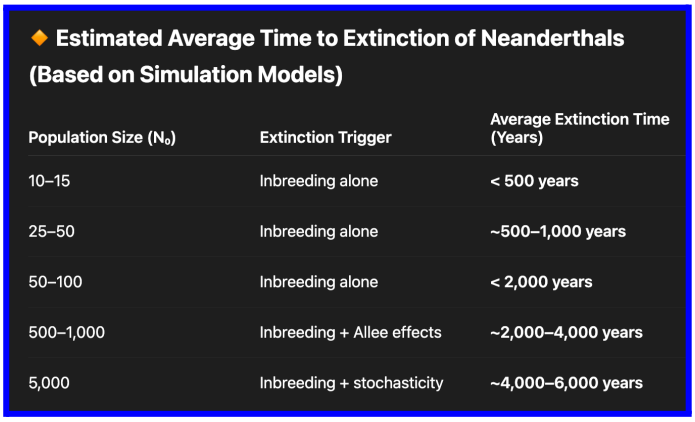
Ten Things Archaeology Tells Us about Neanderthals

"Modern archaeological research has picked away at one of the trickier problems in understanding Neanderthals: How many of them lived together? High-resolution sites (where sediments accumulated slowly and short occupations can be discerned) **confirm that groups likely contained no more than 20 individuals".**

Image 18.

Taking these factors into consideration, population viability models highlight a major inconsistency between empirical demographic limits and evolutionary timelines. When applying the criteria of known population sizes, Neanderthals could not have persisted for even 1,000 years under such conditions, yet radiometric dating frameworks propose that they survived for approximately 400,000 years.

The tension becomes even greater when the same demographic reasoning is extended to *Homo heidelbergensis* and even *Homo erectus*, who are said to have existed for over two million years despite small effective population sizes. This compounds the already well-recognized "hominid fossil gap paradox," which refers to the striking absence of preserved hominid fossils spanning nearly a million years of supposed evolutionary history. Paleoanthropologists acknowledge this as a persistent difficulty, noting that the gap cannot be explained by insufficient exploration or a scarcity of appropriately aged deposits. Instead, the problem lies in the near-total absence of fossils during this interval, with claims of poor preservation or low fossil yield often serving as ad hoc explanations (Gibbons, 2008; Smith, 2018). Similarly, the origin of *Homo* (~2.5–2.0 million years ago) is obscured by the fact that very few fossils clearly document a transition from *Australopithecus* to *Homo habilis* or *Homo erectus*.



🔸 Estimated Average Time to Extinction of Neanderthals (Based on Simulation Models)

Population Size (N_0)	Extinction Trigger	Average Extinction Time (Years)
10–15	Inbreeding alone	< 500 years
25–50	Inbreeding alone	~500–1,000 years
50–100	Inbreeding alone	< 2,000 years
500–1,000	Inbreeding + Allee effects	~2,000–4,000 years
5,000	Inbreeding + stochasticity	~4,000–6,000 years

Image 19. Population size vs extinction time.

The conflict between radiometric dating and genetic evidence highlights a clear inconsistency: one of these frameworks must be incorrect. Genetic data, which are directly measurable, testable, repeatable and predictable, provide a far clearer picture than assumption-based radiometric methods that are known to produce consistent discordant results. For an extended critique of radiometric dating and its limitations, see Nailor, M. (2025), *The Illusion of Deep Time: Systematic Discordant Radiometric Ages and the Myth of an Ancient Ocean Floor*.

When this genetic evidence is considered alongside archaeological and historical data, the convergence becomes even more striking. The earliest known civilizations—encompassing advances in mathematics (Katz, 2009; Robson, 2008), medicine (Nunn, 1996), astronomy (Neugebauer, 1969; Steele, 2007), tool-making and leather tanning (Forbes, 1964), beer brewing (Hornsey, 2003), irrigation (Jacobsen & Adams, 1958; Butzer, 1976), and written records (Cooper, 2004; Michalowski, 1996)—all emerged at the same time only about 5,000 years ago. This timeframe corresponds perfectly with the genetic and demographic evidence previously discussed and provides further confirmation of the young-earth creation (YEC) model.

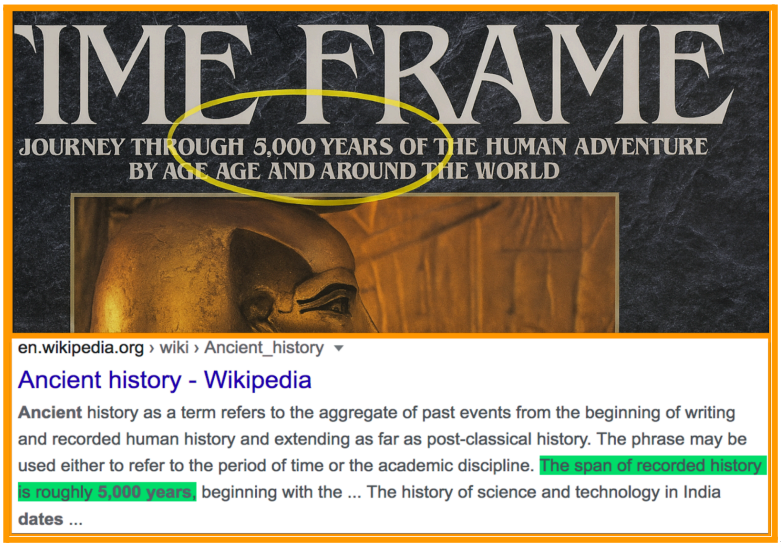
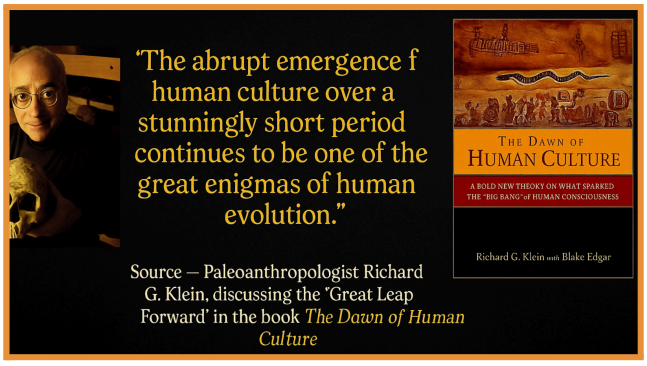


Image 20.

The so-called *Sapient Paradox* remains a major challenge for the evolutionary worldview and will always plague the model. In essence, the paradox highlights that just over 5,000 years ago, there is little to no evidence of mathematics, documentation, irrigation, large populations, organized religion, sophisticated symbolism, writing systems, or other hallmarks of complex society. Then, in a sudden and dramatic shift—described by some scholars as *"the greatest riddle in human history"*—all of these cultural and intellectual capacities appear already fully formed. Rather than gradually developing over tens of thousands of years, the archaeological and historical record shows their near-simultaneous emergence, as though advanced human capacities were abruptly bestowed (see Renfrew, 1996; Mithen, 2007).



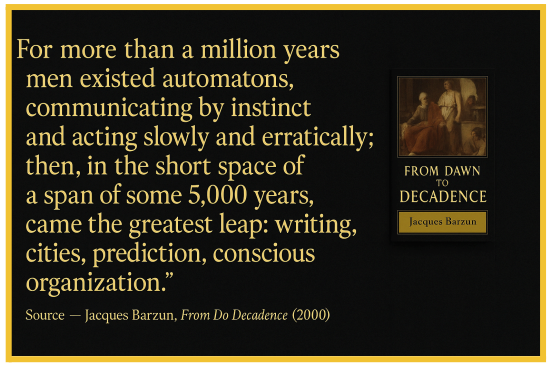


Image 21 & 22 - Quotes about the sapient paradox from two books on human culture & civilizations.

Endogenous Retroviruses (ERVs)

Endogenous retroviruses (ERVs) and related classes of retrotransposons which comprise a substantial portion of mammalian genomes. According to the evolutionary view ERVs are ancient viral sequences that became "stuck" in our DNA when viruses infected the reproductive cells of our ancestors. Instead of disappearing, these viral fragments

eventually became inert and were inherited by each new generation, becoming genetic fossils. In 1969, King and Jukes stated: '99 percent of mammalian DNA is not true genetic material'. This became known as Junk DNA which ERVs became part of after discovery. So while traditionally regarded as genomic fossils and cited as strong evidence for common descent, these elements are increasingly being recognized for their pervasive functionality in gene regulation, embryological development, immune defense, and genome architecture. Primary source studies consistently demonstrate that ERVs are indispensable to normal physiology, with examples including the role of HERV-H in demarcating topologically associating domains in human pluripotent stem cells (Zhang et al., 2019) and their contributions to transcriptional regulation across mammalian genomes (Fueyo et al., 2022; Xiang et al., 2022). These discoveries overturn the "junk DNA" paradigm and call for a reassessment of ERVs as indispensable genomic elements rather than relics of infection.

In early 2000, Linda K. Walkup predicted that transposable elements—including ERVs—serve a functional role. Walkup later published an article about this titled "'Junk' DNA: evolutionary discards or God's tools?" and was published in Journal of Creation (Technical Journal), Volume 14, Number (2), 2000, pages 18–30, where she proposed that such genetic elements were originally designed to facilitate variation function and adaptation within "biblical kinds". Walkup directly proposed that transposable genetic elements—such as ERVs—were intentionally designed by God to facilitate variation (adaptation) within biblical "kinds", and important roles rather than being mere useless relics of evolution. She specifically predicted that some of these elements would serve vital functional roles, contrary to the evolutionary notion of "junk DNA" where evolution had placed ERV's upon their discovery. Todd C. Wood in this same journal proposed renaming transposable elements as "Altruistic Genetic Elements (AGEs)". Further elaboration by Todd was published in a peer-reviewed article titled: *"The AGEing process: Rapid Post-Flood Intrabaraminic Diversification Caused by Altruistic Genetic Elements (AGEs)"*, published in January 2002, Wood elaborated on this model in greater detail.

It was later that very year that the first beneficial function for ERVs was discovered confirming Linda's prediction, as ERV-derived syncytin identified as essential for placental cell fusion. As time went on her predictions have been confirmed even further. After this, in the year 2000 and onward, ERV LTRs were discovered to be repurposed as gene regulatory elements in various tissues. In 2016 ERV element MER41 was shown to regulate immune gene activation via interferon-mediated response. A 2020 review in *Genome Biology* highlights how ERVs are increasingly understood as regulatory elements in embryogenesis and immune responses, and even promising targets in cancer therapy. In 2021 Research showed that the skin microbiome influences ERV expression, suggesting a role in maintaining skin homeostasis and potentially modulating local immunity. In 2023, a study on zebrafish identified an ERV-derived Env38 protein that is interferon-inducible and essential for mounting adaptive immune responses against viral infection.

A 2024 study in mink found that an ERV-V env gene is conserved and highly expressed in testicular tissue, closely linked to seasonal reproductive cycles—indicating a functional role in host physiology. As time goes on more functions are discovered.

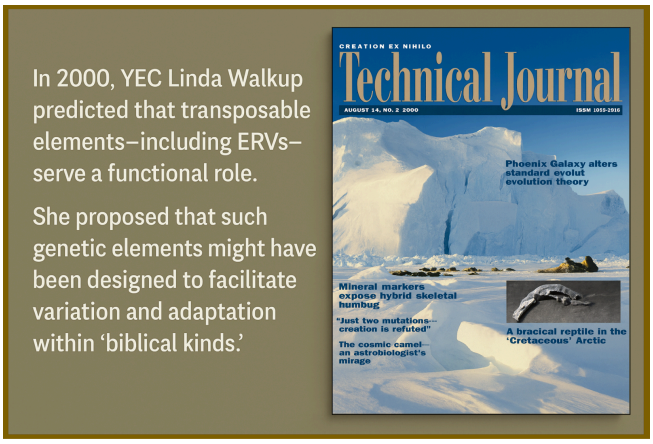


Image 23.

The prevailing evolutionary explanation of "co-option"—wherein non-functional viral remnants allegedly gradually acquire essential regulatory and developmental roles—lacks empirical demonstration and fails to provide testable predictions remaining nothing more than speculative. Instead, the weight of evidence points to intentional integration of ERV-like sequences into genomes and widespread functionality, consistent with a common design framework. For example, ERVs participate in viral mimicry and tumor suppression (Jansz & Faulkner, 2021; Zhou et al., 2021), immune and antiviral defense (Buttler & Chuong, 2022), and stress adaptation. These functions are not rare exceptions but widespread recurrent phenomena. The consistent emergence of function across every identified subclass of transposable elements (LINEs, SINEs, ERVs, DNA transposons) undermines the notion of non-functional "genomic fossils" and better aligns with predictions made by models of designed diversity and genomic archetypes.

Furthermore, arguments for shared ancestry based on ERV distribution and mutational hierarchies in long terminal repeats (LTRs) rest on questionable assumptions. Mutations are not purely random: they cluster in hotspots and are shaped by epigenetic context (Monroe et al., 2022; Melamed et al., 2022). This recognition diminishes the reliability of shared mutations as phylogenetic markers, since parallel patterns can emerge through non-random mutational biases and functional constraints. Additionally, the existence of unfixed ERV insertions in human populations (Marchi et al., 2014) further indicates that not all ERV sequences represent ancient viral remnants, but instead reflect rare cases of endogenization in a fallen genomic landscape. Crucially, fixed ERV sequences consistently show functionality, supporting the creation model's prediction that designed genomic elements are indispensable, while occasional unfixed, deleterious insertions represent genomic corruption in a fallen world.

Contrary to the old "junk DNA" dogma, many endogenous retroviral (ERV) sequences are now known to serve crucial roles in host biology. For example, genome-wide studies confirm that ERV-derived promoters initiate transcription in over one-fifth of the human genome. For example, a genome-wide analysis identified over **51,000 ERV-derived promoter sequences** in the human genome that initiate transcription of nearby genes, often in a tissue-specific fashion (Conley *et al.*, 2008). Likewise, specialized ERV proteins such as syncytins are essential for mammalian placenta formation, mediating trophoblast cell fusion during pregnancy. Other ERV sequences regulate early developmental gene networks and modulate the immune system responses. These findings align with earlier creation-oriented predictions that transposable elements (including ERVs) would have function. In short, many ERV loci exhibit integrative, beneficial functions — not the useless "junk" once assumed and predicted by evolutionists.

From a design perspective, these observations imply that viruses and hosts originated together, and not through later viruses invasion. Indeed, even secular virologists note a paradox: "*viruses need cells, but viruses kill cells*", making virus-first scenarios hard to explain. Instead, as creationist propose an exogenization model: ERVs were present from the beginning and later released infectious particles. Supporting this, intelligent design advocates Liu and Soper observe that the simplest explanation is that *"ERVs with fixed locations and conserved beneficial genes may have been incorporated into the host genome at the time of creation," and that "exogenous retroviruses may have been created to help the ERVs and to transfer useful genes between hosts".* This co-origin view predicts coordinated virus-host systems. Supporting this, modern experiments show interferon (a key antiviral cytokine) activating ERV genes in human cells, and ERV-encoded proteins in turn specifically target the innate immune response (Buttler & Chuong, 2022).. Remarkably, this ERV–immune interplay is found in all mammals tested, suggesting an integrated and conserved design feature. Such irreducibly complex ERV–host networks are difficult to explain by stepwise co-option assembled by chance, but they align naturally with the expectation of a purpose-driven genomic architecture.

The Segregated Design Model (SDM) reinterprets ERVs as endogenous control modules intentionally positioned within the genome. Their "segregated" nature reflects partitioned distribution across chromosomes and populations, consistent with an intelligently organized system of regulated diversity rather than stochastic viral integration. Hierarchical similarity across taxa is viewed as evidence of shared design logic, not ancestry.

Model Foundations

Front-Loaded Functional Diversity. ERVs function as built-in regulatory systems—enhancers, promoters, and non-coding RNAs that permit differential expression without requiring novel mutations (ENCODE Consortium, 2012; Chuong et al., 2016).

Non-Random Localization. ERV loci cluster in transcriptionally active or regulatory regions (Ito et al., 2017), implying purposeful placement.

Segregation Dynamics.

Differences in ERV presence reflect inheritance and recombination rather than infection; fixation equilibrium arises naturally through segregated variation.

Functional Typology

Full-Length ERVs (5′LTR–gag–pol–env–3′LTR) act as multifunctional regulatory cassettes influencing gene silencing and chromatin insulation (Grow et al., 2015). Solo LTRs result from homologous recombination between LTRs, leaving single, stable enhancers—streamlined endpoints optimized for regulation (Badarinarayan & Sauter, 2021).

Population-Level Expression

ERV variation among populations is attributed to segregated inheritance. Fixed ERVs are universal within a kind; unfixed ERVs are polymorphic variants enabling regulatory diversity. Population equilibrium mimics fixation but requires no new insertions.

Testable Predictions of the Design Model

- *ERVs display measurable biochemical activity.*
- *Integration sites correspond to regulatory domains, not random loci.*
- *Population polymorphisms reflect functional segregation.*
- *Redundant regulatory buffering confirms intentional design.*
- *ERVs initiate zygotic genome activation (ZGA) during early embryogenesis—without them, development halts.*
- *ERVs shape the p53 "guardian of the genome" network, critical in preventing cancer.*
- *ERV proteins (e.g., HERV-K Rec) modulate immune responses and protect embryos from infection.*

Prediction: As research progresses, every ERV subclass will be found to have crucial regulatory or structural roles.

Fatal Problems for the "Viral Insertion" Hypothesis

If ERVs were truly the result of thousands of viral infections:

- *Apoptosis Conflict — Viral DNA in germ cells should trigger programmed cell death. Thousands of successful insertions are biologically implausible.*
- *Reproductive Cell Integrity — Germline infections usually destroy fertility, yet ERVs are concentrated in these very cells.*
- *Lack of Proof — No clear exogenous "ancestor virus" matches known ERVs.*
- *Rapid Viral Decay — Modern viruses mutate so quickly that "ancient" ERV preservation is inconsistent with evolutionary timescales.*
- *Cross-Species Contradictions — Near-identical ERVs occur in unrelated animals (cats/baboons, possums/monkeys), contradicting phylogenetic expectations.*
- *Functional persistence, lack of transitional endogenization, and viral mimicry suggest that viruses may be degenerate offshoots of genomic design (Forterre, 2006). The SDM inverts causality: viral forms copy host code, not vice versa.*

Prediction: No laboratory will ever observe a retrovirus going from non-functional to functionally beneficial. This is a prediction we can test and witness in real time since the human HERV-K insertions present a spectrum — some are fixed, while some like HERV-K113 are present in some people but absent in others (Belshaw *et al.*, 2005).

Function Over Fossil: A Design Perspective

ERVs function like normal genes and are expressed in a tissue-specific, stage-specific manner—more consistent with designed regulatory networks than random viral debris. The evidence shows:

- Syncytin genes (ERV-derived envelopes) are essential for placenta formation in both primates and mice, yet arose independently—an impossible "convergent evolution" event.
- ERVs contribute to stress response, immune priming, and antiviral defense—suggesting foresightful programming rather than accident.

- ERVs are consistently integrated into regulatory circuits rather than randomly scattered.

Prediction: ERVs will increasingly be shown to act as “switchboards” coordinating multiple genes in networks, especially during development and immunity.

The Evolutionary Dead-End

Evolutionists invoke “co-option” or “convergent evolution” as their only arguments to explain the precise, functional integration of ERVs. But this begs the question: how could random viral DNA pre-code regulatory networks for complex organisms? The creationist model, by contrast, predicts that ERVs were present from the beginning as functional DNA units, later corrupted by mutation.

Prediction: As sequencing expands, the pattern will show that ERVs sequences will line up with organism-specific regulatory needs.

- New ERV functions will be found in development and physiology. Large-scale genomics already reveals thousands of ERV-derived promoters driving gene expression in development. We expect future studies to uncover even more essential roles for ERVs. For example, ERV sequences have been shown to drive promoters for ~22% of human transcripts , and ERV-derived proteins have been found regulating placental and neural development in mammals. A design hypothesis predicts that additional ERVs will be discovered as key regulators (in brain, immunity, reproduction, etc.), rather than inert relics.
- Modern retroviruses will trace to endogenous origins. If viruses were created with hosts, then any exogenous retrovirus should ultimately derive from the host genome (the exogenization idea). In practice, this means that circulating retroviruses ought to match endogenous sequences in the same “created kinds” of animals. We predict that every pathogenic retrovirus will have a corresponding ERV ancestor in its host. For example, analyses should show that Koala retrovirus (KoRV) and other retroviruses correspond to ERV loci in koalas, reflecting their release from genomic elements. This contrasts with a purely evolutionary view, which expects independent viral invasions; the design prediction is that no retrovirus exists without a genomic “source” in that species.In other words, what we observe as retroviruses in the wild could have ultimately descended from the host’s own genomic elements. This “exogenization” hypothesis predicts a very intimate genetic relationship between modern retroviruses and specific host ERV sequences. In practical terms, **every pathogenic retrovirus is expected to have a close counterpart**

within the host genome from which it could have derived. This is a startling prediction, but there is some evidence consistent with it. For example, the **Koala retrovirus (KoRV)** is currently found as both an exogenous virus infecting koalas and as endogenous proviral inserts in the koala genome — in fact, koalas today are in the midst of an active endogenization process by KoRV (Tarlinton *et al.*, 2006). From an evolutionary standpoint, KoRV is a virus that recently jumped into koalas and is now becoming an inherited part of their DNA. The design perspective can view it conversely: the koala's genome already carried a "dormant" copy of a related retroviral element (or was predisposed to host it), and this element has now produced an infectious form (or at least allowed the virus to proliferate in this species). Notably, all koalas do not carry KoRV insertions in the same genomic locations — some individuals have more copies than others — consistent with a still-spreading infection (Tarlinton *et al.*, 2006). According to the creation model, truly **created (original) ERVs** would differ from these infectious insertions in key ways. They would tend to be present at the same loci in all members of a species (or "kind"), having been part of the initial genome design, whereas infectious insertions will appear randomly and variably across individuals.

- Analogous functions use different ERVs in each lineage. The design model predicts that separate "kinds" were equipped independently with ERVs for similar functions. As one test, compare placental species: indeed, the primate, rodent, and ruminant lineages each use unrelated ERV env genes (syncytins) to mediate placental fusion. Instead of expecting the same virus to infect all these lineages (as evolution might), the creation model predicts each lineage will have its own unique ERVs fulfilling analogous roles. Surveying other vertebrates (e.g. marsupials, monotremes, or new model organisms) should reveal lineage-specific ERV-derived genes for reproduction or development if this pattern holds.

- Disruption of ERV networks will be catastrophic. Because ERV-host systems appear irreducibly complex, perturbing them should cause severe defects. For instance, knocking out host factors that control ERVs should be lethal or disruptive. One test is TRIM28, a gene that represses ERVs by chromatin modification. Experiments in mice show that deleting TRIM28 in early embryos causes ERV expression to "wake up" and ultimately leads to embryonic death. Design predicts many such outcomes: loss of ERV regulators or core ERV elements will derail development or immunity. Conversely, the evolutionary model (with ERVs as neutral leftovers) would not predict so many essential failure points.

- Study: Viral Genome Size Distribution Does not Correlate with the Antiquity of the Host Lineages by José A. Campillo-Balderas et al, *"Our results also suggest that since retroviruses appear to be restricted to plants and vertebrates, they could not have played a role in the evolutionary transition from primitive cellular RNA genomes to the extant DNA-based genetic systems of extant cells, nor the viral reverse transcriptase can be considered an evolutionary vestige of the polymerase that played a role in this transition.*

- *"All known viruses need a cellular host to replicate, thus necessitating the existence of cells before virus survival."* Arshan Nasir et al 2012

Prediction Framework: Created vs. Infectious ERVs

1. Structural Integrity

- *Created ERVs: Expected to be incomplete viral structures — often missing essential viral genes (gag, pol, env) and/or lacking intact LTRs. This cripples the ability to produce infectious particles but leaves behind modular regulatory or structural functions (promoters, enhancers, regulatory RNAs).*
- *Infectious ERVs: Should retain intact gag, pol, and env genes (at least in early insertion stages) and full-length LTRs, making them potentially active or capable of reconstitution into retroviruses.*

Testable Prediction:

- *Systematic genome surveys will reveal that when scientists look closely across the whole genome, they'll find that most of the "functional" endogenous retroviruses (ERVs) that help with development or the immune system aren't actually full viruses — they're just broken pieces of what look like ancient viruses that got stuck in our DNA a long time ago and do not function as viruses at all and never did.*

- *In contrast, pathogenic ERV-like sequences (e.g., KoRV in koalas, HERV-K in humans) will show intact or semi-intact gag/pol/env frameworks, consistent with post-Fall infection events. Reactivating one of these types of ERV's will reawaken its original infection capabilities.*

2. Integration Patterns

- Created ERVs: Should exhibit conserved loci across individuals of a kind, placed in regulatory hotspots (e.g., near developmentally important genes), with evidence of coordinated transcriptional control.
- Infectious ERVs: Expected to insert more randomly (though with some preference for open chromatin), generating polymorphism between individuals and lineages.

Testable Prediction:

- *Created ERVs will appear fixed and highly conserved within a kind's genome.*
- *Infectious ERVs will often appear as polymorphic insertions, with variation in presence/absence among individuals of the same species.*

3. Functional Orientation

- Created ERVs: Predominantly regulatory in role (promoters, enhancers, RNA scaffolds, transcriptional insulators). They will lack evidence of viral particle production.
- Infectious ERVs: Expected to show transcription of viral proteins under certain conditions (e.g., env, gag expression in cancer or immunosuppression).

Testable Prediction:

- Created ERVs will overwhelmingly show regulatory RNA/protein fragments only.
- Infectious ERVs (HERV-K, KoRV) will show intact viral protein expression detectable by proteomics or antibody assays.

4. Cross-Species Comparison

- Created ERVs: Each "created kind" will have unique, kind-specific ERV modules optimized for its biology (e.g., different syncytins in primates vs. ruminants).
- Infectious ERVs: Should show cross-species mobility (e.g., KoRV jumping within marsupials), reflecting post-Fall spread.

Testable Prediction:

- Created ERVs will form non-homologous solutions to the same problem (different ERV env genes co-opted for placenta in different mammals).
- Infectious ERVs will show clear phylogenetic continuity with circulating retroviruses.

Concise PowerPoint Prediction

How to Identify Created vs. Infectious ERVs

- Created ERVs
    - Missing gag, pol, env, or LTRs → non-infectious
    - Conserved, fixed in all members of a kind

    - Function: regulation, development, immunity
    - Lineage-specific, optimized solutions
    - Do not produce viral particles
- Infectious ERVs
    - Retain gag, pol, env, intact LTRs → potentially infectious
    - Often polymorphic between individuals
    - Function: can produce viral proteins/particles
    - Cross-species mobility possible
    - Associated with disease (e.g., HERV-K, KoRV)

Head-to-Head Predictions: ERVs — Evolution vs. Creation

1. Functionality

- Evolution: ERVs = originally predicted as nothing more than pure genomic "junk," non-functional remnants of ancient viral infections.
- Creation: ERVs = predicted in 2000 as functional elements, designed with host, essential roles in development, immunity, and physiology.

2. Origin of Viruses

- Evolution: Viruses emerged independently (virus-first hypothesis), then invaded genomes.
- Creation: Viruses and hosts created simultaneously; viruses cannot exist without a host → escape hypothesis.

3. Exogenous vs. Endogenous Relationship

- Evolution: Exogenous retroviruses infected germlines → became ERVs.
- Creation: ERVs existed first; exogenous retroviruses arise from ERVs (exogenization). Host and ERV created simultaneously.

4. Shared ERVs Across Species

- Evolution: Identical ERVs across species = proof of common ancestry.
- Creation: Shared ERVs = hotspots of insertion preference or common design features; testable prediction: independent but similar ERV integration motifs.

5. Distribution Patterns

- Evolution: ERV insertions random; presence/absence reflects historical infection events.
- Creation: ERV distribution non-random, lineage-specific, and designed for unique regulatory functions (e.g., lineage-specific syncytins).

6. Irreducible Networks

- Evolution: ERV-host interactions co-opted gradually by selection.
- Creation: ERV-host interactions are irreducibly complex, required from the start; knockout experiments should show catastrophic defects.

7. Conservation Across Kinds

- Evolution: Conservation = constraint after infection → some ERVs occasionally co-opted.
- Creation: Conservation = intentional preservation of functional ERVs; predicts all "highly conserved" ERVs are beneficial.

8. Novel Functions

- Evolution: Expect most ERVs to remain non-functional; only rare cases of co-option.
- Creation: Predicts thousands of novel functions will continue to be discovered in ERVs (development, brain, reproduction, immunity).

9. Phylogenetic Patterns

- Evolution: ERVs follow evolutionary trees of common descent .
- Creation: ERVs follow "created kinds"; expect mismatches to evolutionary trees (e.g., ERVs unique to species but absent in supposed ancestors eg HERV-K).

10. Disease Association

- Evolution: ERVs mostly neutral, but will be harmful when activated.
- Creation: ERVs primarily beneficial, with harmful activity resulting from genomic decay from mutation not viral because of corruption post-Fall.

11. Regulatory Roles

- Evolution: Host occasionally recruits ERVs for regulation (exaptation).
- Creation: ERV regulatory roles designed from the start; expect coordinated ERV promoter/enhancer activity across developmental stages.

12. Viral Diversity

- Evolution: Viral diversity accumulates over millions of years from independent origins.
- Creation: Viral diversity originated within kinds; exogenous viral variation arises from corruption/mutation of created ERVs.

13. Host-Specificity

- Evolution: Viral infections cross species over time → ERVs spread into multiple lineages.
- Creation: Each host kind created with unique ERVs tailored to its biology; testable prediction: unique ERV sets per created kind (eg orphan genes).

14. Insertion Hotspots

- Evolution: ERV integration random → shared loci must mean ancestry.
- Creation: ERV integration site hotspots designed and determined by DNA sequence/structure; predict shared insertions without common descent.

15. Future Discoveries

- Evolution: More "dead" ERVs with rare cases of co-option.
- Creation: An ever growing catalog of ERV functions will continue to overturn "junk DNA" assumptions and validate design.

ERVs were initially used as evidence of common ancestry and genomic decay (fossil viruses that no longer function) regarded as worthless junk DNA. But when scientists found some ERVs with important host functions, evolutionists reframed them as "co-opted." To creationists, this is clearly shifting the story and unfalsifiable circular reasoning: "*If it's broken DNA, that proves evolution; if it's functional DNA, that also proves evolution.*" The p53 gene is regulated by ERV Studies have found that p53, is a tumor suppressor protein which binds to specific sequences within the Long Terminal Repeats (LTRs) of certain human ERV subfamilies like LTR10 and MER61.

- ERVs acting as regulatory elements: These ERVs containing p53 binding sites function as active regulatory elements, influencing the expression of genes located nearby, including some known p53 target genes.
- Expanding the p53 network: These ERVs in the genome appear to control the p53 transcriptional network in primates, creating lineage-specific regulatory pathways not found in other species.

- Potential for creating new genes: In one instance, an ERV insertion with a p53 binding site is thought to have played a role in the creation of a new gene, TP53AP1, which is now part of the human-specific p53 regulatory network.

- In some bats this p53 gene had a duplication and now bats are almost cancer free and have extraordinary lifespans. For example the little brown bat (Myotis lucifugus) has two copies of the tumor suppressor gene p53, which contributes to its high resistance to cancer. While many bat species have enhanced p53 activity, Myotis lucifugus specifically possesses a genomic duplication of the TP53 gene. Additionally, some studies indicate that this particular species may even possess up to seven copies of p53, including one full duplication and several shorter retrocopies. Another species, Eptesicus fuscus, appears to have two copies of TP53.

Some ERV envelope proteins expressed in the host can act as decoy receptors or interferents against exogenous viruses — for example, an endogenous retroviral env in mice (Fv4) confers resistance to feline leukemia virus by blocking viral entry, essentially an antiviral defense that was "pre-installed" in the genome (Yan *et al.*, 2009). A design perspective uniquely anticipated such pervasive functionality, whereas the traditional view considered it surprising and had to introduce the concept of "co-option" after the fact to explain each new ERV function discovered.

Nested Hierarchies
Nested ERV distributions parallel phylogenetic trees (Katzourakis & Gifford, 2010). Yet hierarchical reuse of regulatory modules also yields nested patterns which were predicted by creationists. Design systems exhibit tiered modularity analogous to software architecture (Denton, 2016).

Cross-species comparison of ERVs offers another test. The design model holds that each created kind was endowed with its own unique set of ERV elements optimally suited for its biology. Therefore, when we see similar functions across different kinds (e.g., placental fusion in primates vs in ruminants), we expect those functions to be carried out by *different* ERV sequences in each lineage (since each was independently equipped, rather than all inheriting the same viral insertion). This is exactly what we observe with syncytins as discussed — primates, rodents, and other mammals use distinct ERV env genes for placental development (Vernochet *et al.*, 2014). No single retrovirus infected all those lineages to seed the same gene; rather, each lineage has a unique solution. By contrast, truly infectious retroviruses often exhibit cross-species transmission — the same or very similar viruses can be found jumping between host species. If ERVs were solely the products of random infection as evolution poses, we might expect more cases where the *same* ERV sequence is found in very divergent hosts due to horizontal transfer. In reality, ERVs follow the host phylogeny

(Katzourakis & Gifford, 2010) — which evolutionists interpret as insertion in a common ancestor, but creationists interpret and predicted prior as common design or parallel insertion preferences.

Where clear cross-species jumps are known (for example, the gibbon ape leukemia virus is extremely similar to koala retrovirus, hinting that a retrovirus of that group can move between primates and marsupials), those viruses correspond to the **exogenous, infectious category**. The creation prediction would say that no truly *essential* host function will be found to depend on an ERV that is also roaming around as a transmissible virus. Instead, essential functions will be tied to lineage-specific ERV elements.

Retroviruses that infect multiple species (like lentiviruses or gammaretroviruses) do not appear to be foundational to host viability; they are often pathogenic instead. This pattern matches a design expectation that **created ERVs were intended for that host** and stayed mostly within that lineage, whereas **post-Fall viruses can promiscuously spread but are deleterious**.

These specific predictions can be tested by further genomic, developmental, and comparative studies. For example, systematic knockout or knockdown of candidate ERVs should reveal functions in accord with the design model. Comparative genomics can check whether ERV distribution patterns match design (unique, conserved uses) or chance insertion. In all cases, the design framework forces clear, falsifiable hypotheses. If, in future work, ERVs continue to reveal coordinated roles and no truly host-independent viruses are found, that would favor the design interpretation over the conventional story. Each of these design-based predictions is empirically testable: they invite experiments (gene expression profiling, functional knockouts, receptor assays, phylogenetics of viruses and ERVs, etc.) to confirm whether viruses and hosts co-originated. As such, a young-earth design hypothesis for ERVs makes concrete forecasts that can be validated or refuted by data.

Taken together, the evidence refutes the notion that ERVs are best explained by evolutionary leftovers. Instead, ERV-like elements exhibit irreducibly complex associations with gene regulation, development, immunity, and tumor suppression that evolutionary theory has not predicted nor adequately explained. The Biblical creation model, which anticipates treasure rather than junk within the genome, provides a coherent framework that both explains these functional discoveries and makes testable predictions about yet-unknown roles. As more research continues to validate function across transposable element subclasses, the case for design over descent strengthens, challenging one of the evolutionary paradigm's most celebrated arguments.

To summarize

We find consistent differences between ERV-like sequences that appear to be **"original equipment"** in the genome and those that behave like recent viral insertions. Created ERV elements are typically broken as viruses (lacking essential genes), present in all members of a group (conserved loci), enriched in regulatory roles, often unique to one lineage for a given function, and necessary for normal development or physiology. Infectious ERVs, in contrast, tend to be intact or nearly intact viruses, show insertional variability between individuals, can produce viral proteins/particles especially in disease states, sometimes cross species, and are frequently associated with harmful effects (like cancer or immunodeficiency when they are active). These distinctions were **predicted by the design model** and are being borne out as our genomic and virologic knowledge increases. Evolutionary theory can accommodate these facts by post-hoc explanations (for instance, arguing that host genomes eventually domesticated some viral inserts and discarded others), but notably it did not *predict* the sheer extent of functional integration we now see. Early evolutionary geneticists expected most ERVs to be functionless "junk," an expectation decisively falsified by modern functional genomics (Bock & Stoye, 2000). The creation perspective, which from the start suggested ERVs had purpose and were part of an originally good design, is gaining credence as these elements turn out to be far more than genomic fossils.

It is telling that what was once considered a **fatal evidence for common descent** — the nested hierarchies of shared ERVs — are better explained by a design paradigm that employs common design and hotspot insertions. Hierarchical patterns can emerge from designed reuse just as from descent, especially when similar biological problems are solved in different organisms by analogous components (Denton, 2016). The existence of nested ERV distributions is not a smoking gun for unguided evolution; rather, it was a creationist prediction from the start that created design DNA like ERV's will not only show beneficial function but a hierarchical pattern based on function. This is the strength of the Biblical creation model that should challenge the naturalistic secular camp to investigate why those ERVs are there in the first place. Increasingly, we find they are there *for a reason*.

Discussion

The cumulative weight of the evidence surveyed in this paper reveals a striking pattern: evolutionary theory has repeatedly failed in its predictive capacity, while creationist predictions rooted in design-based reasoning have been confirmed time and again. This tension is not a matter of isolated anomalies but of systemic misalignment between evolutionary expectations and empirical reality.

From its inception, Darwinian theory was offered as a scientific alternative to the doctrine of creation. Yet the historical record shows that much of the evidence now heralded as

triumphs of evolution were first predicted by creationist scholars on the basis of design. Ernst Mayr (1963), one of the 20th century's foremost evolutionary biologists, asserted that *"the search for homologous genes is quite futile except in very close relatives,"* believing that random mutations would obliterate similarity across distant taxa. In sharp contrast, Henry Morris (1975) explicitly predicted that homologous genes would be widespread across life due to common design. Subsequent discoveries confirmed Morris, not Mayr. The identification of Hox clusters in Drosophila (Lewis, 1978) and their universal presence across animals (Carroll, 2005, 2018) shocked evolutionary biologists. As Carroll candidly admitted: *"No biologist had even the foggiest notion that such similarities could exist between genes of such different animals"* (2005, pp. 64–65). The fact that such findings were initially regarded as devastating to evolutionary expectations but immediately reframed as triumphs illustrates the plasticity—and thus the unfalsifiability—of the evolutionary framework. As Richard Lewontin once acknowledged, "*For what good is a theory that is guaranteed to agree with all conceivable observations, irrespective of the real structure of the world? ... Is that not exactly the situation with Darwinism? Such a theory can never be falsified*" (1972, p. 181).

This pattern extends far beyond Hox clusters. The supposed "tree of life"—long held as the central visualization of descent with modification—has collapsed under the weight of molecular evidence. Carl Woese, pioneer of molecular systematics, observed that "phylogenetic incongruities can be seen everywhere in the universal tree" (1998). Michael Syvanen (2009) admitted: *"We've just annihilated the tree of life."* W. Ford Doolittle (1999) concluded that the history of life *"cannot properly be represented as a tree,"* while Eric Bapteste summarized bluntly: "*We have NO evidence AT ALL that the tree of life is a reality*" (New Scientist, 2009). Instead of yielding a neat, branching diagram, genes tell contradictory stories; morphology conflicts with molecules; and proteins such as cytochrome b produce *"absurd phylogenies"* (Lee, 1999). The tree, far from being confirmed, has been *"politely buried"* (Rose, 2000). Such results are wholly consistent with a creationist paradigm that expects discontinuity between created kinds, not a seamless web of descent.

The case of the Italian wall lizard (Podarcis sicula) is equally revealing. Evolutionists trumpeted the appearance of cecal valves as the *"evolution of a novel morphological structure on extremely short time scales"* (Herrel et al., 2008). Yet subsequent evidence demonstrated that these valves could disappear within weeks when diet shifted (Vervust et al., 2010), that they are common in related herbivorous species (Sagonas et al., 2015), and that they likely represent latent genetic capacity or epigenetic re-expression (Menton, 2008; Wile, 2012). Far from illustrating the origin of new anatomical information, the lizards exemplify plasticity within pre-existing design. As Stephen Buranyi (2022) observed, plasticity evokes transformations *"you might expect to find in comic books and science fiction,"* but it does not demonstrate the generation of new genetic blueprints. This

pattern matches William Paley's (1802) analogy of common design far better than it does Darwin's imagined ascent of novelty through mutation.

Indeed, the very engine of evolutionary theory—mutation—has failed to deliver the predicted innovation. Experiments with Hox genes in fruit flies yield only malformations, duplications, or losses, never genuinely new, functional structures (Lewis, 1978; Carroll, 2005). So-called "beneficial mutations" across life generally involve degradation—loss of vision in cavefish, tusklessness in elephants, flightlessness in birds—or regulatory shifts of pre-existing traits, not the creation of new anatomy. Even evolutionary insiders acknowledge the crisis. Denis Noble declared: *"In this article, I will show that all the central assumptions of the Modern Synthesis (often also called Neo-Darwinism) have been disproved"* (2013, p. 1235). Lynn Margulis admitted: *"My attempts to demonstrate evolution by an experiment carried on for more than 40 years have completely failed"* (1991). Derek Ager lamented: *"Nearly all the evolutionary stories I learned as a student have now been debunked."* Nils Heribert-Nilsson summarized bluntly: *"The idea of an evolution rests on pure belief."*

Nowhere is the disparity between prediction and observation clearer than in mutation-rate studies. Evolutionary timelines demand slow, steady molecular clocks calibrated by fossils. Yet every pedigree-based study, from Howell et al. (1996) to Parsons et al. (1997, 1998) to Helgason et al. (2024), has revealed mutation rates 10–20 times faster than expected. As Ann Gibbons reported in *Science* (1998): *"Using the new clock, [mitochondrial Eve] would be a mere 6,000 years old."* These results, consistently replicated across humans, chickens, penguins, and other species tested using germline mutations collapse deep time and land squarely within the biblical timeframe. When even the FBI relies on pedigree rates for forensic identification, while evolutionists dismiss them as "problematic," the credibility of evolutionary chronologies is undermined. Hence why Ann Gibbons states; "*Evolutionists are most concerned about the effect of a faster mutation rate.*"

Population-level data reinforce the same conclusion. Founder effects that evolutionists claim should doom populations to extinction have instead produced thriving lineages, such as the Kerguelen mouflon sheep, which exceeded evolutionary predictions of heterozygosity (Kaeuffer et al., 2006). Neanderthals and other hominins, if truly small, isolated groups, could not have persisted for hundreds of thousands of years; demographic models predict extinction within centuries, not millennia. Archaeology compounds the problem: the "Sapient Paradox" highlights that civilization—mathematics, writing, astronomy, irrigation—exploded suddenly only 5,000 years ago (Renfrew, 1996; Mithen, 2007), precisely in line with the biblical record.

Taken together, these lines of evidence converge with remarkable consistency. Genetic similarity across taxa, long assumed to be an evolutionary triumph, was first predicted by creationists and only retroactively adopted by evolutionists after the data forced the issue. The "tree of life" has collapsed under contradictory molecular evidence. Experimental

evolution fails to produce novelty. Beneficial mutations overwhelmingly degrade rather than innovate. Pedigree mutation rates align with biblical timescales, not evolutionary ones. Population bottlenecks, far from being fatal, align with the Flood narrative. Civilization itself arose suddenly and recently, not gradually across deep time.

As Henry Gee (1999) acknowledged, "*To take a line of fossils and claim that they represent a lineage is not a scientific hypothesis that can be tested, but an assertion that carries the same validity as a bedtime story.*" When leading figures within evolutionary biology—Margulis, Noble, Lewontin, Ager, Tahmisian—confess that the theory is unfalsifiable, rests on belief, or has failed experimentally, it should prompt serious re-examination. Michael Ruse, himself an ardent evolutionist, conceded: "Evolution is promulgated as an ideology, a secular religion—a full-fledged alternative to Christianity… This was true of evolution in the beginning, and it is true of evolution still today" (2000).

Just as the supposed creative power of mutation collapses under scrutiny, so too does one of evolution's most celebrated evidences—endogenous retroviruses (ERVs). Once hailed as genomic fossils proving common descent, ERVs are now being revealed as essential, functional elements consistent with design

Endogenous retroviruses (ERVs), once dismissed as "junk DNA" and paraded as evidence of ancient viral infections, are now recognized as indispensable to normal biology. Genome-wide studies reveal that ERV promoters regulate over one-fifth of human genes, syncytin proteins are essential for placental development, and ERV loci shape embryogenesis, immunity, and tumor suppression (Zhang et al., 2019; Fueyo et al., 2022; Xiang et al., 2022). Such discoveries have overturned the evolutionary assumption of useless genomic fossils and confirmed long-standing creationist predictions that transposable elements would prove functional.

In line with design expectations, syncytin ERV genes are absolutely essential for proper placental development. In humans and other primates, the genes **syncytin-1** (from the HERV-W family) and **syncytin-2** (from HERV-FRD) are expressed in the trophoblast and drive the formation of the syncytiotrophoblast layer, which is crucial for nutrient exchange in the placenta. Mice, by contrast, do not have those particular ERVs — but they have their own distinct syncytin-A and syncytin-B genes (from unrelated ERV insertions) that fulfill the same role in rodent placentation. Knocking out syncytin-A in mice causes failed cell fusion at the maternal-fetal interface and embryonic death, underscoring that this ERV-derived gene is vital for development (Dupressoir *et al.*, 2009). The remarkable fact is that **primate and murine syncytins share no common viral origin** — they represent independent capture of different retroviral env genes, yet both are indispensable in their respective lineages (Vernochet *et al.*, 2014). Such an "impossible" convergent acquisition of critical functionality is exactly what a design model would predict: each created "kind" was originally endowed with the ERV genes necessary for placenta formation.

Evolutionary theory, lacking foresight, must invoke extremely improbable coincidence (multiple different retroviral infections all happened to insert an env gene that became essential for placentation in different clades). The patterned distribution of syncytins — found in primates, rodents, lagomorphs, ruminants, carnivores, each from unrelated ERVs — looks more consistent with an intentional design solution repeated in a lineage-specific manner (Vernochet *et al.*, 2014).

Evolutionary explanations rely on **"co-option,"** suggesting that non-functional viral debris somehow became essential regulators. Yet this is not an observed mechanism but a post hoc story. By contrast, a design framework predicts functionality from the outset. The conserved ERV–immune interplay across mammals, the ERV-dependent regulation of the p53 tumor suppressor network, and lineage-specific ERV solutions for placental fusion all display hallmarks of foresight and irreducible complexity. These observations align far more closely with intentional design than with chance viral invasions.

Creationist models further explain why most ERVs lack intact viral genes, appear in conserved regulatory hotspots, and function in organism-specific ways. In this view, ERVs were created as regulatory modules, while modern infectious retroviruses represent corrupted derivatives (exogenization). Testable predictions flow naturally: future research will uncover crucial roles for every ERV subclass, reveal lineage-specific ERVs optimized for unique biological needs, and show catastrophic effects when ERV networks are disrupted. In short, ERVs—once a "smoking gun" for evolution—are now among the strongest evidences for common design.

Conclusion

The theory of evolution, as presently formulated, lacks falsifiability—a foundational pillar of credible scientific theories. A robust scientific framework must generate clear, testable predictions. Yet evolutionary theory falters even at its core mechanism—mutation—by failing to predict or demonstrably produce genuinely novel anatomical structures in experimental or natural contexts. This absence of empirical macroevolution constitutes a profound scientific limitation.

Often cited examples of "beneficial mutations" typically involve minor or degradative changes and do not support large-scale transitions, such as "bacteria to buffalo" or "fish to human." These cases fall short of substantiating the emergence of substantive evolutionary innovation.

Moreover, evolutionary theory enjoys a unique level of legal protection. In *Epperson v. Arkansas* (1968), the U.S. Supreme Court unanimously struck down a law criminalizing the teaching of human evolution, ruling it unconstitutional under the Establishment Clause (Epperson, 1968). Following this, in *Edwards v. Aguillard* (1987), the Court invalidated a

Louisiana statute requiring “creation science” to accompany evolutionary instruction, citing its clear religious intent and violation of constitutional separation of church and state (Edwards, 1987). These precedents underscore that evolution is unparalleled among scientific theories in its explicit protection through constitutional law.

Compounding these issues is the explicit reticence among proponents to engage with dissenting evidence. As one critic noted, evolutionary biologists have been accused of dismissing out-of-context quotes that challenge foundational assumptions (NCSE, 2015). Moreover, Dr. Yvonne Boldt (2019) asserted that *“a growing contingent of scientists … find the evidence for Darwinian evolution wanting”* (Boldt, 2019). Joe Felsenstein has observed that *“random mutations most certainly degrade the genome, never sophisticate it”* (Felsenstein, 2020). Eugene McCarthy who discovered that the phylogenetic tree that forms from genetics looking at cytochrome C shows that pigs follow chimpanzees, breaking the nested hierarchical pattern. His hypothesis was rejected, and why? “His manuscript was submitted to Oxford University Press but declined due to lack of scientific consensus”. Imagine that, rejected for no other reason than it goes against mainstream views. He said… "IF YOU CRITICIZE DARWIN, IT'S LIKE SAYING THERE IS NO JESUS IN A BAPTIST CHURCH." In an interview titled Did we come from pigs? We read “Despite all the advancements in the understanding of evolution, McCarthy thinks some of the rhetoric around neo-Darwinism amounts to dogma.”These statements suggest that acknowledging and critically engaging with contradictory evidence remains problematic within segments of the field.

Image 24.

Some are so bold to even admit “*materialism is absolute, we cannot allow a divine foot in the door*" - Richard Lewontin, Harvard Geneticist.

> *Richard C. Lewontin Evolutionary Biologist, Harvard University "For what good is a theory that is guaranteed … to agree with all conceivable observations, irrespective of the real structure of the world? …Is that not exactly the situation with Darwinism?* ***Such a theory can never be falsified.***"

> Source: Lewontin, "Testing the Theory of Natural Selection," Nature, Mar. 24, 1972, p. 181.

In sum, the combination of an unfalsifiable core, absence of documented macroevolutionary innovation, exceptional legal protection stacked with the Freedom From Religion Foundation who actively sue individuals who criticise evolution theory, and selective dismissal of inconvenient evidence raises fundamental questions about the empirical and epistemological foundations of evolutionary theory.

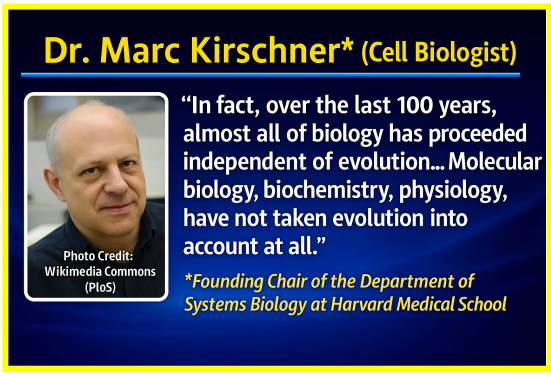
> *"It must be significant that nearly all the evolutionary stories I learned as a student.. have now been debunked"* - Dr. Derek V. Ager, Department of Geology, Imperial College, London

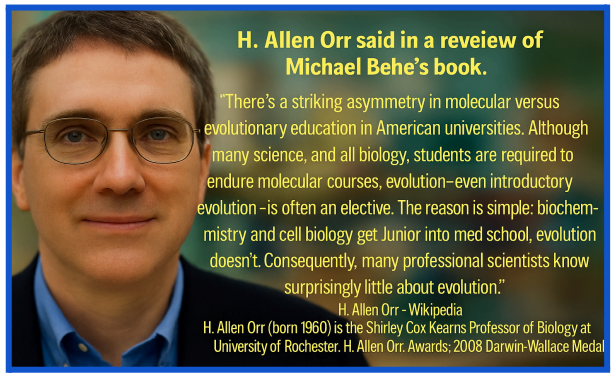
The role of evolutionary theory is to explain the diversity of life on earth. It has failed at doing this in multiple ways. The explanatory power of the model is negated by its failed predictions and ignoring observable data simply because it contradicts the evolutionary narrative.

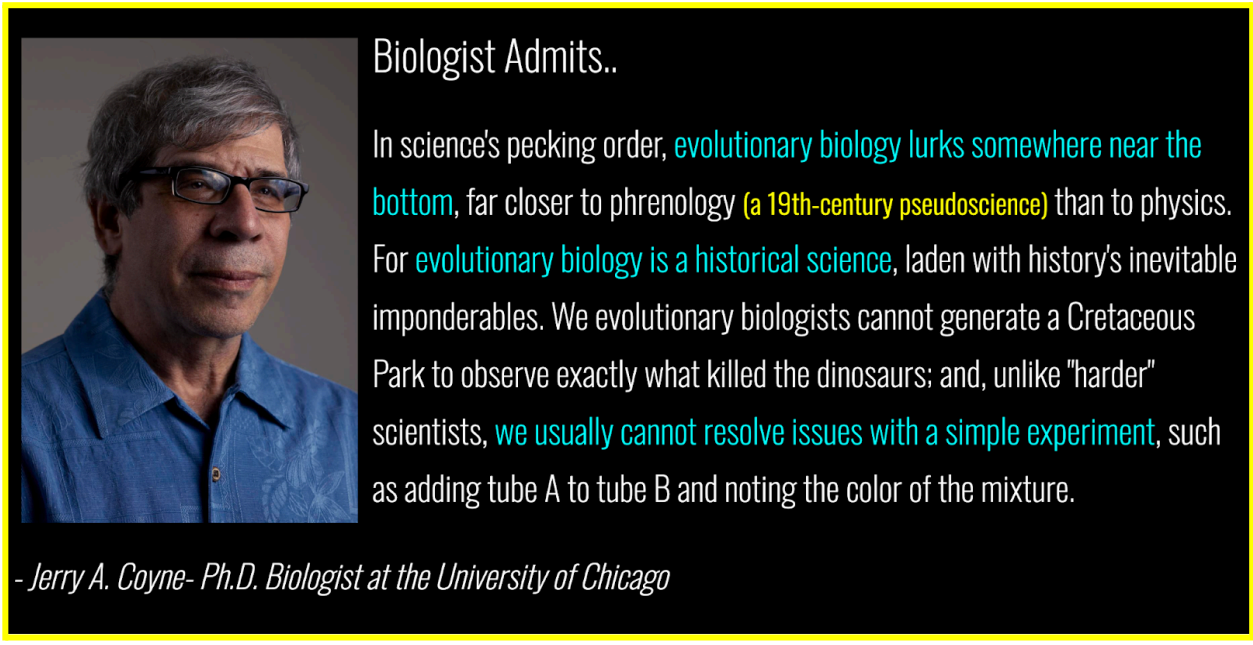
The creation model has demonstrated genuine predictive power. From Linnaeus and Owen to Morris and modern baraminologists, creationists anticipated patterns of similarity, genetic clustering, population recovery, mutation rates, and limited variation within kinds. These predictions, repeatedly borne out by data, affirm that design-based reasoning offers a coherent, testable, and falsifiable framework for understanding life.

In the end, the central question is not whether organisms change—they clearly do—but whether those changes ever generate genuinely new, complex anatomical structures through mutation and selection. The evidence reviewed here provides a consistent answer: no. As Dr. Richard Bliss former professor of biology and science education at Christian Heritage College once wrote, "*The miracles required to make evolution feasible are far greater in number and far harder to believe than the miracle of creation.*"

The time has come, therefore, to acknowledge what the data demands. The central assumptions of the Modern Synthesis have been disproved. Evolutionary theory, propped up by ad hoc explanations and ideological commitment, has lost its explanatory power. By contrast, the design framework—rooted in Scripture, affirmed by history, and vindicated by empirical evidence—stands as the better scientific model.







"The final prediction of this study is that defenders of evolutionism will dismiss it without reading, either making up pathetic excuses outside of science or using Ai to scan it for them looking for any arguments they can use. While some will even go a step further and fall back on the usual shallow line: 'Where's your Nobel Prize?'"

References

Books & chapters

- Behe, M. J. (2019). *Darwin devolves: The new science about DNA that challenges evolution.* HarperOne.

- Brand, L. (1997). *Faith, reason, and Earth history.* Andrews University Press.
- Carroll, S. B. (2005). *Endless forms most beautiful.* W. W. Norton & Company. https://sandymaguire.me/books/carroll-sean-b-endless-forms-most-beautiful.html
- Collins, F. (2006). *The language of God.* Free Press.
- Darwin, C. (1872). *On the origin of species by means of natural selection* (6th ed.). John Murray.
- Darwin, C. (1838). *Notebook entry on reading Charles Lyell's* Principles of Geology. (Manuscript note).
- De Beer, G. (1969). Introduction. In R. Chambers, *Vestiges of the natural history of creation* (1969 reprint, p. 11).
- Forbes, R. J. (1964). *Studies in ancient technology* (Vol. IV). Brill.
- Gee, H. (1999). *In search of deep time: Beyond the fossil record to a new history of life.* Cornell University Press.
- Hornsey, I. S. (2003). *A history of beer and brewing.* Royal Society of Chemistry.
- Jeanson, N. J. (2017). *Replacing Darwin: The new origin of species.* Master Books.
- Jeanson, N. J. (2022). *Traced: Human DNA's big surprise.* Master Books.
- Katz, V. J. (2009). *A history of mathematics: An introduction.* Addison-Wesley.
- Macrone, M. (1994). *Eureka!* Barnes & Noble.
- Margulis, L. (1970). *Origin of eukaryotic cells.* Yale University Press.
- Margulis, L. (1993). *Symbiosis in cell evolution* (2nd ed.). W. H. Freeman.
- Marsh, F. L. (1941). *Fundamental biology.* Union College Press.
- Marsh, F. L. (1944). *Evolution, creation, and science.* Review and Herald Publishing.
- Marsh, F. L. (1947). *Evolution or special creation?* Eerdmans.
- Martin, W., & Müller, M. (1998). The hydrogen hypothesis for the first eukaryote. In *Nature* (pp. 37–41). Nature Publishing Group.
- Maynard Smith, J., & Szathmáry, E. (1995). *The major transitions in evolution.* Oxford University Press.
- Meyer, S. C. (2013). *Darwin's doubt: The explosive origin of animal life and the case for intelligent design.* HarperOne.
- Mayr, E. 1963. Animal Species and Evolution. Cambridge, MA: Harvard University Press, 609.

- Morris, H. 1975. The Troubled Waters of Evolution. San Diego, CA: Creation-Life Publishers, 84-85

- Michalowski, P. (1996). Mesopotamian cuneiform: Origin. In P. T. Daniels & W. Bright (Eds.), *The world's writing systems* (pp. 159–168). Oxford University Press.

- Morris, H. (1975). *The troubled waters of evolution.* Creation-Life Publishers.

- Morris, H. M. (2012). *Scientific creationism* (Reprint of 1974 ed.). Institute for Creation Research.

- Neugebauer, O. (1969). *The exact sciences in antiquity.* Dover.

- Nunn, J. F. (1996). *Ancient Egyptian medicine.* University of Oklahoma Press.

- Paley, W. (1802). *Natural theology: Or, Evidences of the existence and attributes of the Deity, collected from the appearances of nature* (2nd ed.). R. Faulder.

- Renfrew, C., & Morley, I. (Eds.). (2007). *Becoming human: Innovation in prehistoric material and spiritual culture.* Cambridge University Press. (Includes: Mithen, S., "The Sapient paradox," pp. 21–48.)

- Robson, E. (2008). *Mathematics in ancient Iraq: A social history.* Princeton University Press.

- Steele, J. M. (2007). *Observations and predictions of eclipse times by early astronomers.* Springer.

Journal articles & reviews

- Alexander, M., Ruan, J., Miller, M., Papoulas, O., van Tuinen, M., Glenn, T. C., Reed, K. M., & Mindell, D. P. (2015). Mitogenomic analysis of a 50-generation chicken pedigree reveals a rapid rate of mitochondrial evolution and evidence for paternal mtDNA inheritance. *Biology Letters, 11*(11), 20150702. https://doi.org/10.1098/rsbl.2015.0702

- Alexander, A., Ruan, J., Miller, M., Papoulas, O., & Mindell, D. P. (2013). The evolution of mitochondrial control region and evidence for structural constraints in *Gallus gallus*. *Molecular Biology and Evolution, 30*(8), 1752–1765.

- Arakaki, Y., et al. (2013). The simplest integrated multicellular organism unveiled. *PLoS ONE, 8*(12), e81641. https://doi.org/10.1371/journal.pone.0081641

- Bapteste, E. (2009). Why Darwin was wrong about the tree of life. *New Scientist* (Magazine article).

- Bedford, T., Cobey, S., & Pascual, M. (2011). Strength and tempo of selection revealed in viral gene genealogies. *BMC Evolutionary Biology, 11*, 220. https://doi.org/10.1186/1471-2148-11-220

- Booth, A., & Doolittle, W. F. (2015). Eukaryogenesis, how special really? *Proceedings of the National Academy of Sciences, 112*(33), 10278–10285.

https://doi.org/10.1073/pnas.1421376112

- Brandenburger, C., et al. (2019). Rapid radiation in angiosperms. *Nature Communications, 10*, 422. https://doi.org/10.1038/s41467-019-08353-9
- Buswell, J. M., et al. (2010). Rapid adaptive radiation in plants. *Molecular Ecology, 19*(7), 1381–1393.
- Carroll, S. B. (2005). (See book above.)
- Cavalier-Smith, T. (2002). The phagotrophic origin of eukaryotes and phylogenetic classification of Protozoa. *International Journal of Systematic and Evolutionary Microbiology, 52*(2), 297–354. https://doi.org/10.1099/00207713-52-2-297
- Chen, P., & Zhang, J. (2020). Environment-dependent epistasis mitigates the cost of gene loss during genome reduction. *Genome Biology and Evolution, 12*(6), 747–761. https://doi.org/10.1093/gbe/evaa093
- Crandall, K. A., et al. (2012). Calibrating the molecular clock in crustaceans. *Molecular Phylogenetics and Evolution, 62*(2), 329–344. (DOI not provided)
- Dávalos, L. M., et al. (2012). Molecular phylogenetics of bats. *Biological Journal of the Linnean Society.* (Full article title/DOI not provided)
- Darlington, C. D. (1959). The origin of Darwinism. *Scientific American, 200*(5), 60–66.
- Doolittle, W. F. (1999). Phylogenetic classification and the universal tree. *Science, 284*(5423), 2124–2129. https://doi.org/10.1126/science.284.5423.2124
- Dyer, E. E., Cassey, P., Redding, D. W., Collen, B., Franks, V., Gaston, K. J., Jones, K. E., Kark, S., Orme, C. D. L., & Blackburn, T. M. (2017). The global distribution and drivers of alien bird species richness. *PLOS Biology, 15*(1), e2000942. https://doi.org/10.1371/journal.pbio.2000942
- Fan, H., et al. (2018). Rapid speciation in birds. *Science, 360*(6393), 1442–1446. (DOI not provided)
- Feschotte, C. (2008). Transposable elements and the evolution of regulatory networks. *Nature Reviews Genetics, 9*, 397–405. https://doi.org/10.1038/nrg2337
- Galtier, N., et al. (2008). Incongruence between gene trees. *Trends in Ecology & Evolution, 23*(9), 482–487. https://doi.org/10.1016/j.tree.2008.03.017
- Gibbons, A. (1998). Calibrating the mitochondrial clock. *Science, 279*(5347), 28–29. https://doi.org/10.1126/science.279.5347.28
- Gould, S. J. (1979). Darwin vindicated! *New York Review of Books, 26*(1), 36–38.
- Gray, M. W. (2012). Mitochondrial evolution. *Cold Spring Harbor Perspectives in Biology, 4*(9), a011403. https://doi.org/10.1101/cshperspect.a011403

- Grow, E. J., et al. (2020). Endogenous retroviruses function as species-specific enhancers of stem cell pluripotency. *Genome Biology, 21*, 288. https://doi.org/10.1186/s13059-020-02134-9
- Gura, T. (2000). Bones, molecules, or both? *Nature, 406*, 230. https://doi.org/10.1038/35018664
- Hajibabaei, M., Singer, G. A. C., Hebert, P. D. N., & Hickey, D. A. (2006). DNA barcodes distinguish animal species. *PLoS Biology, 4*(10), e422. https://doi.org/10.1371/journal.pbio.0040422
- Hartl, D. L., & Clark, A. G. (2007). *Principles of population genetics* (Book; 4th ed.). Sinauer.
- Hebert, P. D. N., Cywinska, A., Ball, S. L., & deWaard, J. R. (2003/2004b). Biological identifications through DNA barcodes. *Proceedings of the Royal Society B, 270*(1512), 313–321. https://doi.org/10.1098/rspb.2002.2218
- Helgason, A., Árnadóttir, E. R., Moore, K. H. S., Guðmundsdóttir, V. B., Ebenesersdóttir, S. S., Guity, K., Jónsson, H., & Stefánsson, K. (2024). The rate and nature of mitochondrial DNA mutations in human pedigrees. *Cell, 187*(15), 3904–3918.e8.
- Helgason, A., Einarsson, A. W., Guðbjartsson, D. F., & Stefánsson, K. (2015). The Y-chromosome point mutation rate in humans. *Nature Genetics, 47*(5), 453–457. https://doi.org/10.1038/ng.3216
- Hendry, A. (2009). Evolutionary biology: Lizards in an evolutionary lab. (University/McGill commentary; no DOI).
- Herrel, A., Huyghe, K., Vanhooydonck, B., Backeljau, T., Breugelmans, K., Grbac, I., Van Damme, R., & Irschick, D. J. (2008). Rapid large-scale evolutionary divergence in morphology and performance associated with exploitation of a different dietary resource. *Proceedings of the National Academy of Sciences, 105*(12), 4792–4795. https://doi.org/10.1073/pnas.0711998105
- Howell, N., Elson, J. L., Turnbull, D. M., & Herrnstadt, C. (1996). How rapidly does the human mitochondrial genome evolve? *American Journal of Human Genetics, 59*(3), 501–509. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1914922/
- Howell, N., Elson, J. L., Herrnstadt, C., Shults, C., & Turnbull, D. M. (2003). The pedigree rate of sequence divergence in the human mitochondrial genome: There is a difference between phylogenetic and pedigree rates. *American Journal of Human Genetics, 72*(3), 659–670. https://doi.org/10.1086/368264
- Hudson, C. M., et al. (2020). Rapid reptile diversification. *Evolution, 74*(5), 1030–1042.
- Jaenisch, R., & Bird, A. (2003). Epigenetic regulation of gene expression: How the genome integrates intrinsic and environmental signals. *Nature Genetics, 33*, 245–254. https://doi.org/10.1038/ng1089
- Jacobsen, T., & Adams, R. M. (1958). Salt and silt in ancient Mesopotamian agriculture. *Science, 128*(3334), 1251–1258. https://doi.org/10.1126/science.128.3334.1251

- Karmin, M., Saag, L., Vicente, M., Wilson Sayres, M. A., Järve, M., Talas, U. G., Rootsi, S., Ilumäe, A. M., Mägi, R., Mitt, M., Pagani, L., … Metspalu, M. (2015). A recent bottleneck of Y chromosome diversity coincides with a global change in culture. *Genome Research, 25*(4), 459–466. https://doi.org/10.1101/gr.186684.114

- Laland, K. N., Uller, T., Feldman, M. W., Sterelny, K., Müller, G. B., Moczek, A., Jablonka, E., & Odling-Smee, J. (2014). Does evolutionary theory need a rethink? *Nature, 514*(7521), 161–164. https://doi.org/10.1038/514161a

- Lavialle, C., et al. (2021). The role of endogenous retroviruses in development and disease: A review. *Genome Biology, 22*, 158. https://doi.org/10.1186/s13059-021-02366-y

- Lawton, G. (2009, January 21). Why Darwin was wrong about the tree of life. *New Scientist.* (Magazine article)

- Lee, M. S. Y. (1999). Molecular phylogenetics of mammals. *Trends in Ecology & Evolution, 14*(3), 110–117. https://doi.org/10.1016/S0169-5347(98)01567-0

- Leslie, M. (2001). Into the limelight. *Science, 294*(5549), 2059. (News item)

- Marshall, C. R. (2016). Explaining the Cambrian "explosion" of animals. *Annual Review of Earth and Planetary Sciences, 44*, 335–355. https://doi.org/10.1146/annurev-earth-060115-012428

- Martin, W. F., Garg, S., & Zimorski, V. (2015). Endosymbiotic theories for eukaryote origin. *Philosophical Transactions of the Royal Society B, 370*(1678), 20140330. https://doi.org/10.1098/rstb.2014.0330

- Maeda, R. K., et al. (2009). The bithorax complex of *Drosophila*: An exceptional Hox cluster. *Advances in Genetics*. https://doi.org/10.1016/S0070-2153(09)88001-0

- Mi, S., Lee, X., Li, X., et al. (2000). Syncytin is a captive retroviral envelope protein involved in human placental morphogenesis. *Nature, 403*(6771), 785–789. https://doi.org/10.1038/35001608

- Nabholz, B., Glemin, S., & Galtier, N. (2009). The erratic mitochondrial clock: Variations of mutation rate, not population size, affect mtDNA diversity across birds and mammals. *BMC Evolutionary Biology, 9*, 54. https://doi.org/10.1186/1471-2148-9-54

- Nilsson, D.-E. (2009). The evolution of eyes and visually guided behaviour. *Philosophical Transactions of the Royal Society B, 364*(1531), 2833–2847. https://doi.org/10.1098/rstb.2009.0082

- Nowack, E. C. M., & Melkonian, M. (2010). Endosymbiotic associations within protists. *Philosophical Transactions of the Royal Society B, 365*(1541), 699–712. https://doi.org/10.1098/rstb.2009.0188

- Parsons, T. J., Muniec, D. S., Sullivan, K., Woodyatt, N., Alliston-Greiner, R., Wilson, M. R., Berry, D. L., Holland, K. A., Weedn, V. W., Gill, P., & Holland, M. M. (1997). A high observed substitution rate in the human mitochondrial DNA control region. *Nature Genetics,*

15(4), 363–368. https://doi.org/10.1038/ng0497-363

- Pisco, A. O., Brock, A., Zhou, J., Moor, A., Mojtahedi, M., Jackson, D., & Huang, S. (2018). Non-Darwinian dynamics in therapy-induced cancer drug resistance. *Proceedings of the National Academy of Sciences, 115*(48), 12271–12276. https://doi.org/10.1073/pnas.1814189115

- Prochnik, S. E., et al. (2010). Genomic analysis of organismal complexity in the multicellular green alga *Volvox carteri*. *Science, 329*(5988), 223–226. https://doi.org/10.1126/science.1188800

- Renfrew, C. (1996). The sapient paradox: Can we explain the enigma of the human revolution? *Cambridge Archaeological Journal, 6*(1), 3–7. https://doi.org/10.1017/S0959774300001419

- Rokas, A., & Carroll, S. B. (2006). Bushes in the tree of life. *PLoS Biology, 4*(11), e352. https://doi.org/10.1371/journal.pbio.0040352

- Sagan, L. (1967). On the origin of mitosing cells. *Journal of Theoretical Biology, 14*(3), 225–274. https://doi.org/10.1016/0022-5193(67)90079-3

- Sagonas, K., Pafilis, P., & Valakos, E. D. (2015). The impact of insularity on dietary niche variation of a Mediterranean lizard. *Scientific Reports, 5*, 14193. https://doi.org/10.1038/srep14193

- Sigurdardottir, S., Helgason, A., Gulcher, J. R., Stefansson, K., & Donnelly, P. (2000). The mutation rate in the human mtDNA control region. *American Journal of Human Genetics, 66*(5), 1599–1609. https://doi.org/10.1086/302844

- Starck, J. M. (1999). Phenotypic flexibility of the avian gizzard: Rapid, reversible and repeated changes of organ size in response to changes in diet. *Journal of Experimental Biology, 202*, 3171–3179.

- Stuart, Y. E., et al. (2014). Rapid adaptive divergence. *Proceedings of the National Academy of Sciences, 111*(7), 2093–2098. https://doi.org/10.1073/pnas.1318886111

- Tallavaara, M., Eronen, J. T., & Luoto, M. (2021). Productivity, biodiversity, and pathogens influence the global hunter-gatherer population density. *Proceedings of the National Academy of Sciences, 118*(2), e2018856118. https://doi.org/10.1073/pnas.2018856118

- Tennessen, J. A., Bigham, A. W., O'Connor, T. D., Fu, W., Kenny, E. E., Gravel, S., … Akey, J. M. (2012). Evolution and functional impact of rare coding variation from deep sequencing of human exomes. *Science, 337*(6090), 64–69. https://doi.org/10.1126/science.1219240

- Vervust, B., Van Dongen, S., Grbac, I., & Van Damme, R. (2010). The mystery of the missing cecal valves: Rapid, diet-induced phenotypic plasticity in lizards. *Naturwissenschaften, 97*, 787–793. https://doi.org/10.1007/s00114-010-0692-1

- Ward, R. D., Zemlak, T. S., Innes, B. H., Last, P. R., & Hebert, P. D. N. (2005). DNA barcoding Australia's fish species. *Philosophical Transactions of the Royal Society B, 360*(1462), 1847–1857. https://doi.org/10.1098/rstb.2005.1716

- Willmer, P. (2013). Evolution of animal body plans. *Evolution & Development, 15*(4), 267–271. https://doi.org/10.1111/ede.12039
- Woese, C. (1998). The universal ancestor. *Proceedings of the National Academy of Sciences, 95*(12), 6854–6859. https://doi.org/10.1073/pnas.95.12.6854
- Zhang, X., et al. (2023). An interferon-inducible endogenous retroviral envelope protein is required for adaptive immunity in zebrafish. *PLOS Pathogens, 19*(2), e1011222. https://doi.org/10.1371/journal.ppat.1011222

Legal cases

- *Epperson v. Arkansas*, 393 U.S. 97 (1968).
- *Edwards v. Aguillard*, 482 U.S. 578 (1987).

News, magazines, web items, and commentaries

- Anthropology News. (2018, March 29). Ten things archaeology tells us about Neanderthals. https://www.anthropology-news.org/articles/ten-things-archaeology-tells-us-about-neanderthals/
- BBC Future. (2016). Could just two people repopulate Earth?
- CNN. (2022). How one man's gift led to Australia's feral rabbit invasion.
- Current Conservation. (2021). Edward Blyth: The unsung pioneer of natural selection.
- Darwin Online (University of Cambridge). (2023). Edward Blyth's contributions to natural history.
- National Cancer Institute (SEER). (2020). Mendel's Accountant: Genetic simulation resources. https://surveillance.cancer.gov/genetic-simulation-resources/packages/mendels-accountant/
- National Geographic. (2008, April 21). Lizards evolve new organs. https://www.nationalgeographic.com
- National Geographic. (2020). Bison on Catalina Island and the conservation debate.
- NCSE. (2015). Misquoted scientists respond.
- Science. (2024, January 12). Some crustaceans have evolved a way to make silk. (News item)
- ScienceDaily. (2008, April 17). Lizards evolve in response to new environment. https://www.sciencedaily.com/releases/2008/04/080417112433.htm
- Smithsonian / AMNH Press Office. (2016). New study doubles the estimate of bird species in the world. (Press release)
- Soodyall, H., et al. (2003). The genetic prehistory of southern Africa: Bushmen and the Khoisan peoples. *American Journal of Human Genetics.* (Citation noted; full details not provided

here.)

- Sun, Z., et al. (2024). Endogenous retrovirus V envelope gene expression in mink testis and its association with seasonal reproduction. *Frontiers in Cellular and Infection Microbiology, 14*, 1404431. https://doi.org/10.3389/fcimb.2024.1404431

- Vaughn, M. (2011, August). Charles Darwin and the pleasure of gambling. *Wired Magazine.*

- Wikipedia contributors. (2023). *Mitochondrial Eve.* https://en.wikipedia.org/wiki/Mitochondrial_Eve

- Wolf, M., et al. (2022). Genomic impact of whaling in North Atlantic fin whales. (Provide full journal details if you want a complete APA entry.)

- YLE News. (2018). From a single deer to thousands.

- (Wolves population note). *Rescue of a severely bottlenecked wolf population by a single immigrant.* (Course page link provided; add bibliographic details if a peer-reviewed source is preferred.)

- Science vol 289, p 2342 https://www.newscientist.com/article/dn30-running-slow/

Open-access items you linked

- Herrel, A., et al. (2008). Rapid large-scale evolutionary divergence… *PNAS, 105*(12), 4792–4795. https://doi.org/10.1073/pnas.0711998105 — Full text: https://pmc.ncbi.nlm.nih.gov/articles/PMC2290806/

- Kaeuffer, R., et al. (2007). Unexpected heterozygosity in an island mouflon population founded by a single pair of individuals. *Proceedings of the Royal Society B, 274*(1612), 2495–2501. https://pmc.ncbi.nlm.nih.gov/articles/PMC1766376/

- K. T. NishantThe Baker's Yeast Diploid Genome Is Remarkably Stable in Vegetative Growth and Meiosis

Web essays / blogs / organizational pages

- Paley, R. (2002). An introduction to baraminology. Objectiveministries.org. https://objectiveministries.org/creation/baraminology.html

- Menton, D. (2008). Lizard tales: A closer look at the so-called "new organs." Answers in Genesis.
- **University of Chicago Medical Center. (2012, May 23).** *Human-like spine morphology found in aquatic eel fossil.* Phys.org. https://phys.org/news/2012-05-human-like-spine-morphology-aquatic-eel.html

- Wile, J. (2010/2012). Rapid changes in lizards: Evolution or design? Blog.drwile.com

- Nailor, M. (2025). *One species, many names: Mitochondrial evidence unites humans, Neanderthals, Denisovans, and Heidelbergensis.* https://doi.org/10.5281/zenodo.16936057
- Nailor, M. (2025). *When barcodes blur: Mitochondrial DNA barcoding of Felidae indicates two ancestral lineages?* https://doi.org/10.5281/zenodo.16937646
- Nailor, M. (2025). *The mystery of the missing mutations in plant DNA: Evidence of recent bottlenecks in nightshades.* https://doi.org/10.5281/zenodo.16938026
- Nailor, M. (2025). *Post-Flood populations: Haplogroup formation and fixation dynamics from Noah to Babel dispersion.* https://doi.org/10.5281/zenodo.16938125
- Nailor, M. (2025). *The illusion of deep time: Systematic discordant radiometric ages and the myth of an ancient ocean floor.* https://doi.org/10.5281/zenodo.16938048
- Nailor, M. (2025). When Facts Falsify Fossils: Pedigree Substitution Rates Upend the Evolutionary Timeline.
- University of Chicago Medical Center. (2012, May 23). *Human-like spine morphology found in aquatic eel fossil.* Phys.org. Retrieved [Access Date], from [URL]
- Denton, M. (2016). Evolution: Still a Theory in Crisis. Discovery Institute Press.

Court, opinion, and quote placeholders (non-scholarly — format only if you need them in the bibliography):

- Boldt, Y. (2019). Statement quoted (Microbiology, University of Minnesota).
- Felsenstein, J. (2020). Commentary on mutation claims.
- Rose, M. R. (2009). Quoted in *New Scientist*.
- Syvanen, M. (2009, January 21). Quoted in *New Scientist*.