JOURNAL PAPER

When Facts Falsify Fossils:
Pedigree Substitution Rates Upend the Evolutionary Timeline

By Matt Nailor & Donny Budinsky

Truth In Research (2025)

Abstract

For over half a century, the molecular clock has been calibrated by assumption rather than observation. Since Zuckerkandl and Pauling first proposed a clock-like rate of molecular change (1962), evolutionary geneticists have relied on phylogenetic calibration, tying substitution rates to fossil-based divergence times, most notably the postulated 6–7 million year split between humans and chimpanzees. When this failed, they began calibrating the mutation rate clocks by anchoring them to known historical events, circular reasoning at its finest. In contrast, direct pedigree-based observations tell a radically different story. When mitochondrial DNA is directly measured in known family lineages rather than inferred from fossils, the resulting empirical substitution rates are orders of magnitude faster than those used in evolutionary models. These real-time rates, derived from observed pedigrees, render the slow deep time molecular clock untenable. We cover multiple pedigree studies over decades of time including conducting our own coalescence and fixation analysis as well as a Bayesian coalescent calculation using the average mean observed substitution rate across multiple pedigree studies *(1 substitution every 34.48 generations)* and we included the Biblical history timeline scenario as the litmus test for a fixation rate. The results reveal a Mitochondrial *Eve* lived approximately 6,000–7,200 years ago, precisely aligning with the historical window long preserved in Biblical chronology. Despite this, every online search for *"the age of humanity"* or *"mitochondrial Eve"* by the unsuspecting public, continues to yield evolutionary phylogenetic, fossil-calibrated estimated ages derived from assumptions rather than the factual data. The pedigree evidence, reproducible across independent studies and diverse populations in different species, directly falsifies the fossil record's deep-time dates. It demonstrates that the true molecular clock runs far faster and points unmistakably to a recent, unified origin and bottleneck for all humans and all life. This study redefines not only the timescale of all living species but human ancestry as well, replacing hypothetical evolutionary divergence with measured generational change. In doing so, it restores the mitochondrial clock to what it was always meant to be: a record of real time, not imagined ages.

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.

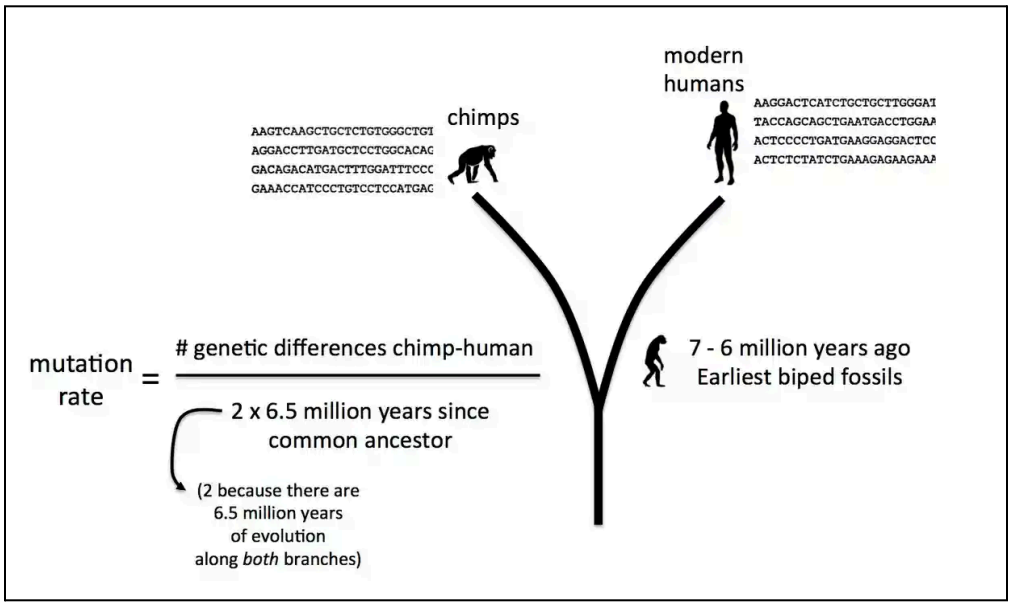


Figure 1. Phylogenetic mutation rate calculation based on assumed ancestor split 6 - 7 million years ago.

*"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.

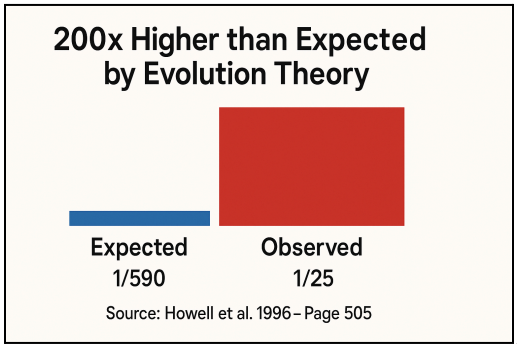
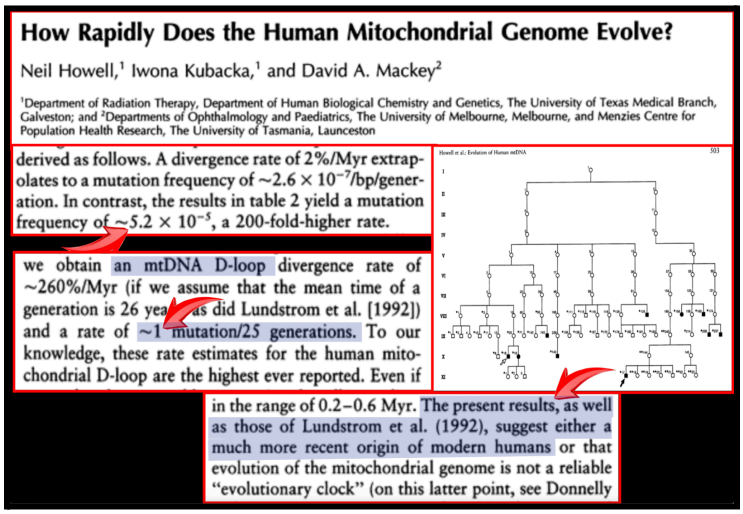


Figure 2. Observed vs Evolutionary Estimated rates.

Observed Mutation Rate


How Rapidly Does the Human Mitochondrial Genome Evolve?

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

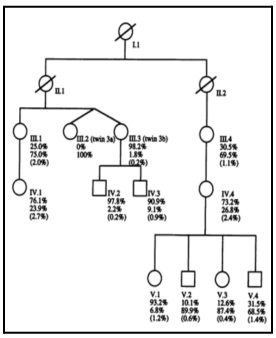
[1]Department of Radiation Therapy, Department of Human Biological Chemistry and Genetics, The University of Texas Medical Branch, Galveston; and [2]Departments of Ophthalmology and Paediatrics, The University of Melbourne, Melbourne, and Menzies Centre for Population Health Research, The University 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. They observed both fast rates and slow rates so they published the average, giving them 1 substitution every 33 generations.

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.

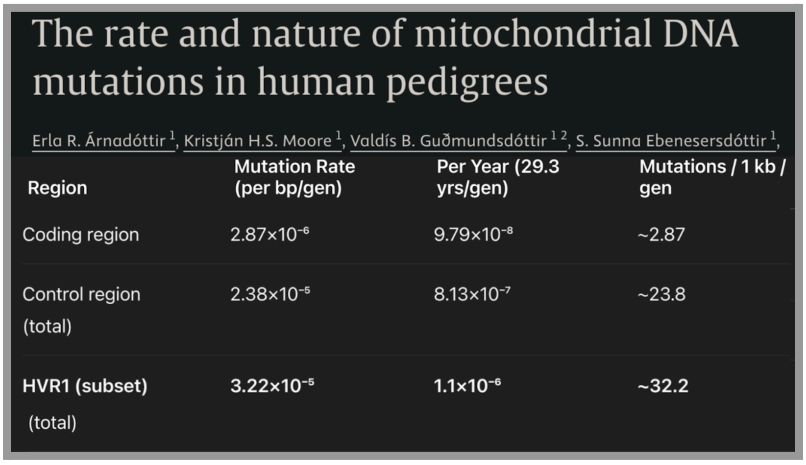
Instead, geneticists look at the best areas in the mtDNA with the weakest selection to obtain the most accurate clock. These regions are called hypervariable regions, specifically HVR1, HVR2.

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 stay the same. From single generation dyad studies to multi-generational pedigrees, the rate is always the same. This tells us the hypervariable regions are functionally neutral with no selection or background selection taking place and the D-Loop is a close second. Therefore invoking selection of any kind to slow this clock down enough to match evolutionary timelines 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.

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 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.

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 –* $\mathbf{2.47 \times 10^{-5}}$*)* 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.

MRCA - Biblical Eve

Now, let's run our own analysis using all of these high quality pedigree studies to obtain the MRCA since now we have published data on the max diversity in the D-Loop, specifically in HVR1 (360 bp) & HVR2 (315 bp) - (B. Budowle 1999);"*The average number of nucleotide differences between individuals in a database is greatest for the African American and African samples (14.1 and 13.1, respectively), and the least variable are the Caucasians (ranging from 7.2 to 8.4)".*

Looking across studies that tested the most diverse people groups for the optimal population wide substitution rates, we find a range from **averages** in diverse studies of 1/25 (*Howell 1996)* 1/30 (*Mumm 1997*), 1/45 (*Bendall 1996*), *1/33 (Parsons 1997), 1/30 (Parsons 1998),* 1/27 (Brandstätter 2004), *1/29 (Santos 2005), 1/39 (Madrigal 2012),, and 1/40-45 (*Árnadóttir & Helgason 2024). This rounds to an average substitution rate of 1 substitution arising every 33 - 34.48 generations). We will use the high end mean below.

Coalescence calculation

> Substitution rate of 0.029 per HVR1+HVR2 locus per generation *(4.29×10^{-5}/site/gen if 676 bp)* = 1 mutation every 34.48 generations 34.48 generations/mutations × 7 mutations (14.1 differences between 2 lineages) = 241 generations 241 generations × 25 - 30 year generation time, average 27 = **6,507 years** *(assuming a 25–30 year generation time, the MRCA falls between 6,025 - 7,230).* To independently check this substitution-rate estimate, I then applied a Bayesian coalescent model *(below)*, an orthogonal method using the Biblical scenario, which converged on basically the same result. We obtained a mean **259 generations** for mitochondrial Eve. Using a 25 year generation time we land on 6,475 years ago for Eve.

This agreement across two fundamentally different inferential approaches *(analytic rate calculation and Bayesian coalescent modeling)* adds substantial confidence to the MRCA estimate. The fact that both the analytic substitution-rate method and the Bayesian coalescent model independently converge on the same 6.5 kyr timescale provides extremely strong corroboration.

Bayesian coalescent

Using the Biblical timeline and a global bottleneck, we will use the hypervariable regions since they make the best lock. Locus: HVR1+HVR2, L = 676 bp *(because 14.1 max differences are for both HVR1+HVR2)*. We applied a Poisson-based coalescence model, assuming neutrality across 95% of hyper variable regions one and two sites, this results in a substitution rate of 0.029/locus/generation. For the Per-locus rate you use: $k_{locus/gen}$= 0.029 locus ′gen 1 (μ = 4.29 × 10^{-5} $site^{-1}gen^{-1}$ i.e.
Link d to t: d ~ Poisson(λ), λ = $2k_{locus/gen}{}^{t}$. With d = 14, the posterior for t has **mean 259 generations** and (95% Crl 145–405 generations (25–30 y/gen = 3.6–12.1 kya).

A recent severe bottleneck shortens expected coalescent times; rapid recent growth has little effect on the TMRCA itself. Including a "bottleneck at ~ 2 kyr post-origin, then expansion" but the posterior remains broad because coalescence is a random process and when a population explodes in size, you get many more copies of the existing lineages, but they all descend from the same handful of mothers who passed through the bottleneck. That doesn't tighten the date, it just gives you more recent duplicates of the same few branches.

Fixation Rate

Next we are going to obtain a fixation rate testing the biblical timeline. We will be using Helgason et al 2024 published mutation rate of the HVR1 region since we know the max diversity and the fact that purifying selection across these regions is virtually zero. Basically all hypervariable sites behave neutrally. Since the substitution rate equals the fixation rate for neutral sites, it seems pointless to run a simulation but lets do it anyway for those who really want to see the numbers.

Since we know the max diversity in both HVR regions; Max diversity = 14.1 *(HVR1+HVR2 combined)*, and the population average = 8.2 *(HVR1+HVR2 combined).* We can use Heyer's observation of HVR hot spots to determine how many bp in each region ticks and how much does not, her limit shows that both hotspots are far below max saturation with **HVR1 (360 bp) at 7.54 fixed substitutions** and **HVR2 (313 bp) 6.56 fixed substitutions**. For the population average we see **HVR1 with 4.39 fixed substitutions** and **HVR2 with 3.81 fixed substitutions.**

We will now run the biblical history for a fixation rate. According to scripture Creation began and Adam and Eve were created around 6,000 *(Masoretic text)* to 7,500 *(Septuagint)* years ago. The family branched into two groups since Cain was outcast. Over 1,500 - 2,500 years later there was a global flood population bottleneck *(4,500 - 5,500 years ago)* that reduced the population of Noah and his family. These groups each grew in their own populations over 600 - 900 years till they unified at the tower of Babel then shortly after broke into 70 groups and spread around the earth; Ham′s line to Africa. This population stayed in smaller tribes and never progressed into agriculture, staying hunting and gathering. This is why the African population ended up having almost twice as many fixed substitutions as the average population world wide (8), since the others took advantage of agriculture and exploded in population growth making substitutions much harder to fixate. The max

fixed differences in HVR1 (360 bp) is 7.54 fixed substitutions and in HVR2 (310 bp) there are 6.56 for a total of 14.1.

Based on this, what would the fixation rate be and does everything line up based on this mutation rate?

Math

Note: The symbol μ (mutation) represents the per-site mutation rate per generation in population-genetic equations.

- HVR1 length L = 360 bp
- Average pedigree mutation rate: M = 5 x 10^{-5} per bp per generation
- Divergence of present-day lineages since the Flood: 4,000–6,000 years ago
- Generation time g ~ 25 years -> t = 160–240 generations since the split

Under neutrality, the substitution (fixation) rate per site equals the mutation rate . The expected pairwise differences between two lineages that split t generations ago is: E[d]′ = 2 ut L

What the Parsons rate predicts

For t = 160–240 and L = 360:

- dexpected = 2 × 5 - 10^{-5} × t × 360
- t = 160 = d ~ 5.76 substitutions in HVR1
- t = 240 = d ~ 8.64 substitutions in HVR1

Your reported values vs. the prediction.

Max observed = 7.54 differences -> sits nicely inside the 5.76–8.64 band.

The " fixation (substitution) rate";
You can invert the formula to estimate the per-site per-generation substitution rate that would produce a given pairwise distance d: $\mu = d/L$ divided by 2t

From the max (7.54): μ = 6.5 × 10^{-5} (for t = 160) down to 4.36 × 10^{-5} (for t = 240). Centered right around the fixation value of 5 × 10^{-5}

Does it "line up" with μ = 5 × 10^{-5}?
Results: Using your biblical timeline (split ~160–240 generations) and ’ μ, the predicted HVR1 pairwise distance range is between 5.76–8.64. Your max of 7.54 fits that prediction perfectly. Exactly the rate Parsons et al. (1997) observed for HVR1. This requires a 25 year generation time however. To obtain the **average** 4.39 fixed substitutions in the population the generation time needs to be shifted up to 35 years. Then the numbers fit perfectly; implied μ: 3.56–5.34 × 10^{-5} per bp per gen. Range; 4.11 – 6.17 substitutions.

Across multiple independent pedigree datasets spanning three decades, the human mtDNA control-region substitution rates done on diverse people groups consistently centers around (4 – 6) × 10^{-5} per site per generation, with a 20–25-yr generation time gives 1.6–3.0×10^{-6} per site per year. So with a typical 2 - 2.5% sequence divergence in either HVR1 or Both HVR1+2. This yields a MRCA age between 3,300 - 7,800 years under realistic generation intervals, a remarkably stable empirical signal.

Using the **average** substitution rate observed from pooled pedigree studies that tested the most diverse people groups including deep rooted pedigrees. We land on Biblical Eve living somewhere between 6,025 - 7,230 years ago using a range of 20 - 30 year generation time. Perfectly in line with predictions and exactly the opposite of evolutionary expectations. Looking at fossil dates in the geologic column to try and alter the observable data just so it agrees with evolutionary views is more proof of evolution's unfalsifiability and pseudoscientific position in science. The reality is, evolution requires mutation rates to be slow and they are the exact opposite in all life. Since evolution is based on mutations and it cannot even get that right, what does that tell you about the rest of the theory? This is why mutation rates are so vital to understand, since the goal of evolution theory is to explain the diversity of life on earth and it cannot do that with the observable data, rather the creationist model can and does. This tips the scales in our favor and places us with the better explanatory and predictive model. This is also why YEC makes predictions using mutation rates and evolutionists cannot. They fail down the line because without anchoring a mutation rate to a historical date or using evolutionary phylogenetic divergence assumptions, they cannot get a slow clock.

This is one reason why no one takes Dr. Nathaniel Jeanson from Answers in Genesis up on his direct head to head pedigree prediction challenge, which is to choose any animal species and make predictions on its mutation rate of any successive generations. Critics know the results will only make them look worse, deep down they know the truth. Another thing to consider is mutation saturation. Think about it, if only 19 different bp regions can mutate in HVR1 and 22 in HVR2 *(Heyer 2001)* and we only have a total of 14 fixed differences in these regions with their fast rate of change, why have not all 40 bp regions all reached fixation over the supposed deep evolutionary timeline? This lack of change, yet rapid rate is contradictory and why it's a paradox. We see this in animal species as well and lack of genetic differences are what prompts evolutionists to ask questions about it like; *"It is thought we split from a common ancestor with chimps 5-6 million years ago,* ***more than enough time for substantial genetic differences to develop****."* - Dr David Whitehouse BBC News Article When Humans Faced Extinction. We creationists know why the evidence is missing, but evolutionists cannot explain it and never will.

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, $k = \mu$, 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.

Neanderthal & Denisovan

Creationists have tried to explain the high mutation differences we found in Neanderthal and Denisovan by hypermutation (wood 2012). We will pose a new concept today that will explain their high nucleotide differences (210 Neanderthal, 385 Denisovan) without invoking a change in the mutation rate at all. You see, in 2018 David Thaler et al discovered that when a new group formed, say a new dog breed or new wild species, independent from another group. That there was a large leap in genetic differences between the two. He explained it like this in an interview by Marlowe Hood *"And 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."*

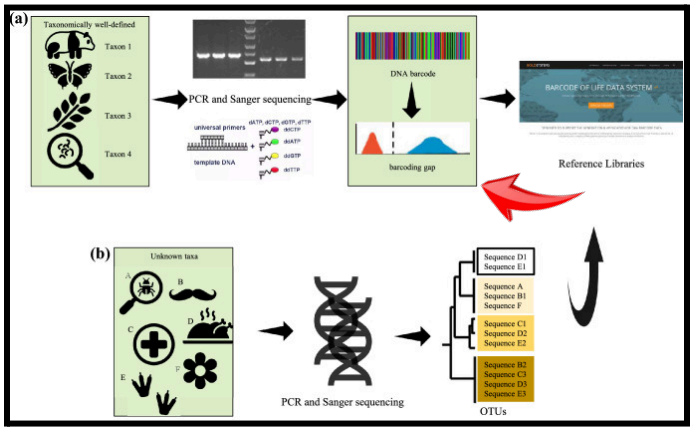


Image 1. The red arrow points to the DNA barcode gap we find between species and subspecies. Image was taken from study titled "Life barcoded by DNA barcodes" by Mali Guo

This “gap” is a mutational leap that occurs in a burst when a new species, subspecies, hybrid, domestication or distinct population emerges. There is no need to invoke hypermutation when we see this in real time. Take for example the modern day canis family. Each new species and subspecies is divided by a large mutational gap, with nothing in between connecting them even though the mutation rate stays the same. Let’s go to the DNA barcoding website BOLDSYSTEMS. We are going to obtain the lowest mutational differences in the database and show how there is a gap between each species.

Before we get into the details, let’s use a simple example of how this works. We can all agree that domestic dogs came from wolves. Yet when we test canis familiaris to canis lupus we find a large genetic boundary “gap” of many mutational differences. No rapid hyper mutation rate, no chain of mutations linking them to each other. This same principle is applied to all life and we can use this observation to explain neanderthal and Denisovan diversity without invoking hypermutation or changing the mutation rate at all actually.

Everyone would agree that chimpanzees and bonobos are related. At some point in the past the bonobo species arose from a speciation event from a chimp. Yet there is a large gap of differences between the two with nothing in between. This massive gap arose like an explosion of mutations that arise out of nowhere. Saltationism (from *saltare*, Latin for *to jump*) is the evolutionary idea that: New species or major biological changes can arise suddenly through large, discontinuous genetic shifts, rather than through many tiny gradual mutations.

Geneticists contemplated such ideas before genetic boundaries were ever discovered, they referred to it as genomic revolutions during speciation. Modern genomics confirms: chromosome changes, genome duplications, and hyper bursts of mutations that can cause rapid speciation and hybridization, sometimes dubbed adaptive radiation which refers to a burst of extremely fast diversification in which one ancestral species quickly splits into many descendant species.

Chimp and bonobo differences within the COI gene fragment are 23 bp different from one another in the COI 650 bp region. This massive difference exists with no link to the chimp, a large gap

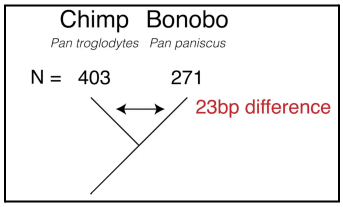


Figure 8. Shows the base pair difference between chimp and bonobo.

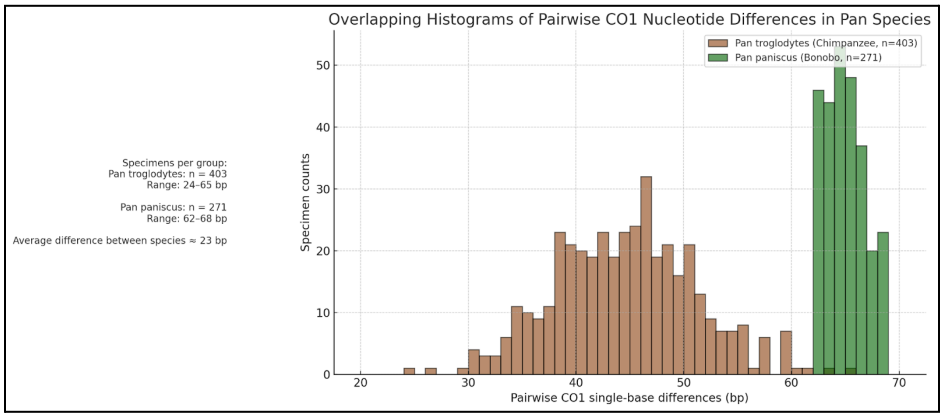


Figure 9. Overlapping histograms were generated in a widescreen format to visualize the distribution of pairwise sequence differences among biologically meaningful pan species groups. Mutation-difference values were plotted for each species, with the number of individuals indicated at left: Pan triglydotes (chimpanzee) 403 species in the database (base pair range 24 - 65).. Pan Paniscus (bonobo), 271 species in the database (base pair range 62 - 68). Average pairwise difference 23 base pairs different. (N = number of species).

So within pan species we find when looking at the 650 bp COI gene fragment that there are 24 differences between the two. Pan mutates much faster than humans in this region, not a single pan species ever tested have even below 24 mutations, yet all hominins have below 20 bp differences with most having 0 mutational differences.

This example found in chimps and bonobos applies to hominin as well. We see most humans with only 0-1 base pair differences between us, while Neanderthals are 7 - 9 bp average away and Denisovan are 17 - 18bp. Since pan species are much more diverse and have mutated much more than humans, their differences reflect that in the "gap". Meaning, the 23 differences we see between pan - equates to the 7 - 10 bp differences we see between hominins in our tree.

This tells us that when one of these new founder groups spawned from another, there was a new burst of diversity that arose instantly. A "gap" that can be seen even today, as the subspecies branched off. So we have Homo sapiens who the Neanderthal branched off of created an 7 - 9 bp gap, then Denisovan branched off Neanderthal creating another 8 - 10 bp gap from them, equaling a total of 17 - 18 bp gap from homo sapiens. This alignment allows us to make a prediction based on differences we see today in the entire mtDNA.

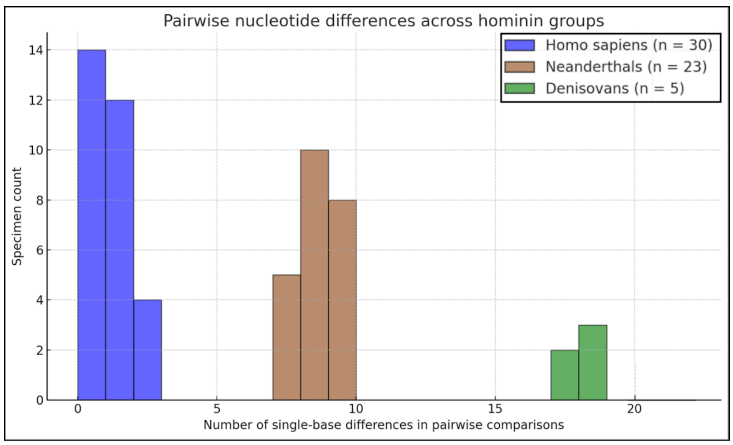


Figure 8. Overlapping histograms of pairwise nucleotide differences across hominin groups. This figure shows how many genetic differences occur within and between Homo sapiens (n = 14), Neanderthals (n = 10), Denisovans (n = 5). The y-axis indicates specimen counts, while the x-axis shows the number of single-base differences in pairwise comparisons. Most Homo sapiens cluster at very low differences (0–1),. Neanderthals peak around (7 - 9) mutations, while Denisovans (17 - 18). All COI barcode sequences obtained from https://v3.boldsystems.org/index.php/TaxBrowser Home

Without altering the mutation rate at all, we predict that we can explain the diversity simply through the arrival of these new groups. So now let's test this prediction. With Neanderthals having 7 - 9 differences away from humans in the 650 bp region, this is 1.08% - 1.38%. Now we need to apply this to the entire mtDNA and see if the total equals the amount of differences we see in Neanderthals.

> 1.08% of 16,569 bp is 179 nucleotide differences
>
> 123% of 16,569 bp is 204 nucleotide differences
>
> 1.38% of 16,569 = 229 nucleotide differences

This is precisely in line with what we observe in Neanderthal differences. Let's follow this trend for testing Denisovan and see if the prediction holds. So from a range of 179 - 229 differences, the arrival of Denisovan from Neanderthal should cause a new gap of differences of the same, a range of 1.08% and 1.38% or 358 - 458 nucleotide differences. This range captures the nucleotide differences. They have 385 perfectly.

The confirmation of this prediction is statistically supported by the levels of diversity seen in both Neanderthals and Denisovans. This is saltationism in action and explains the data perfectly with no alteration of the mutation rate whatsoever.

Since we know that none of these small people groups could have even lived for over 2,000 years based on their small population size (15-20), inbreeding would have killed them *(Krist Vaesen et al 2019)* before that duration. Therefore we want to be able to explain the mutational differences within that window without invoking unobservable scenarios as much as possible and nothing does that better than this observed data we see in DNA barcoding in living species today.

Critics

One argument made against these dates is that the MRCA is not the first woman, she is just one of many. However just using evolutions on criteria based on the population 6,000 years ago refutes this rescue device. Approximately 14 million people lived on Earth 6,000 years ago based on historical population data compiled by organizations like the Population Reference Bureau (PRB) and Gapminder. The numbers only get worse in other studies like Joseph Caspermeyer 2013 who states that the human population shot up from an estimated 4-6 million people to 60-70 million by 4,000 B.C. Now, let's go with the low conservative number because it still won't matter at all. Imagine a world like 6,000 years ago, without technology and only sub par sea navigation like it would have been. People are living in isolated geographic groups *(e.g. islands or continents)* with very limited gene flow between groups and zero migration to/from certain places, e.g. Australia, Andaman Islands *(Indian Ocean)*, Nicobar Islands, Hokkaido & Northern Japan, The Aleutian and Alaskan Interior, Northern Siberia, Tasmania, Papua New Guinea Highlands, etc.... Now in 6,000 years *(230 or so generations)* from now in the future, with that starting population of 14 million people *(about 7 million female*s) alive right now. We want to assess the probability that only one of the 7 million distinct maternal lineages alive today survives to become the MRCA in 6,000 years from now. In other words, would a single present-day woman become the sole mitochondrial DNA most recent common ancestor of everyone 6,000 years hence? This would mean all other current female lineages (mtDNA lines) alive right now go extinct, leaving only one "Eve" from today as the common maternal ancestor of the future populations. Conclusion: "Virtually Zero" Probability. In quantitative terms, the probability is so vanishingly small that for practical purposes it can be considered 0%. Absent an unforeseen global catastrophe that wipes out nearly all but one maternal line globally, we expect tens or hundreds of thousands of distinct mtDNA lineages to still be around in 6,000 - 7,000 years ago, not just one and there is no known bottleneck in the last 7,000 years to account for such a scenario. Think about it, Australian aboriginals have supposedly been in Australia for at least ~50,000 years, with some suggesting 60,000+ years, and for most of that time external influence and major migrations were basically non-existent. So how come when they are tested they also show the exact same low genetic diversity and a shared MRCA along with everyone else? Something does not add up and that is the story of evolution, genetics tells us the truth. The reality is, if a traveler came along and married an aboriginal and had kids, the rest of the entire population would have to die so that sole female child's mtDNA took over to become the MRCA that also just so happens to land on the Biblical Eve timeline. This irrational scenario is all they have to argue. The reality is, any new introduction would not remove the older MRCA from the population until something bottlenecked or diverged the population. The fact is, if there was zero migration between even one of these global regions, then no single woman in one region can ever become the maternal ancestor of people in another isolated region. The math does not lie, the evolutionary story does. With strict isolation, different regions end up with different mitochondrial MRCA women, one per region. There is no way for the world to share a single mtDNA MRCA only 6,000 years after your starting point, because the lineages in, say, Australia and the Andaman Islands never mix. But let's see simulation results because talk is cheap.

I also ran a quick Monte Carlo simulation with a smaller but similar setup, just to get an intuitive feel:

- Population of N_f = 7,000 women instead of 7,000,000 (1/1000th the size, so coalescence should be *much faster* than in the real case).
- Generations: 230.
- Each generation, every woman chooses her mother randomly from the previous generation (a standard Wright–Fisher maternal model).
- Start with 7,000 unique maternal lineages (one per woman).
- Ask: after 230 generations, how many distinct maternal lineages are left?

Result (20 separate runs):

- After 230 generations, the number of surviving lineages was always between 55 and 69, with an average of about 62.
- Not even once did the simulation end with only 1 surviving lineage.
- And remember: this was with 7,000 women, not 7,000,000. With the real population size, coalescence is even slower, so you'd keep **far more** than 60 maternal lines.

So in a tiny, highly simplified world, you still have dozens of independent maternal lines after 6,000 years. Scaling up to the real world only strengthens the point: you don't get down to 1.

Let's start with the most generous case for rapid coalescence with all people together:

- No subgroups, no islands, no isolation.
- Every generation, each person's mother is drawn randomly from the 7 million women of the previous generation.
- Population size stays roughly **N_f = 7,000,000 women**.

In standard population genetics for a mitochondrial (haploid) genome, the typical time to a single maternal MRCA is on the order of the female population size in generations.

- Very simple rule of thumb: Expected time to a single mtDNA MRCA ≈ N_f generations.
- So with N_f = 7,000,000 women: `Expected MRCA time ≈ 7,000,000 generations.`
- If you assume 25 years per generation: `7,000,000 generations × 25 years ≈ 175,000,000 years.`

Obviously we don't have that much human history, this just tells you that with such a huge population, you cannot expect everyone's mtDNA to funnel down to one woman in only 230 generations.

A simple approximation for the probability that we hit the MRCA within `t` generations is:

```
P(MRCA within t generations) ≈ t / N_f
```

Plug in your numbers: $t = 230 \quad N_f = 7{,}000{,}000 \quad P \approx 230 / 7{,}000{,}000$

Which is: `P ≈ 0.000033` (about a 0.0033% chance to have a single mtDNA lineage)

And that's already with the very generous assumption of one giant, well-mixed population. Once you add real-world structure, islands, and possible isolated groups, it gets *much* worse.

If you start with millions of women spread across partly isolated regions, there is no realistic way that only one of those women becomes the direct maternal ancestor of everyone just 6,000 years later. Under standard evolutionary math, the mtDNA MRCA for such a big, structured population would be much, much older than 6,000 years, or you'd end up with multiple regional MRCAs, not one global "Eve". But we do not have to stop there, let's REALLY try to help evolution out and drop the female population to a segregated 1,000 all at the same place and same time.

I ran **2,000 independent simulations** with:

- `N_f = 1000`
- `generations = 230`

Results:

- Minimum number of surviving original lines: 4
- Maximum: 15
- Average: about 8.9
- Median: 9
- Number of runs where only 1 lineage survived: 0 out of 2,000

After 6,000 years, you still have around 9 distinct maternal lineages on average.
In 2,000 trials, you **never** end up with just **one** surviving lineage.

That means the probability of everything collapsing to a single "Eve" in only 230 generations is less than 1 / 2000 ≈ 0.0005 (and very likely much smaller).

Remember: this is the most favorable possible case for a rapid single MRCA:

- Tiny population (1,000 women, not millions).
- Perfect mixing (no geographic isolation, no separate continents).
- Constant population size (no nasty bottlenecks that kill people unevenly).

Even then, after just 6,000 years you don't get one maternal ancestor — you get several. Sorry evolution, you cannot get it to work at all. Invoking the MRCA is not the first when the dates consistently land on recent dates in human history only makes things worse. This rescue device is put to rest.

Critics have also tried to argue that we cherry pick studies and ignore a few that show a slow rate of change. They say this because they do not understand the topic and are looking at studies that are not

even trying to obtain a population wide substitution rate for a MRCA, they just point to studies that are single relation and often shallow depth pedigree and low depth NGS detection to observe mutations studies where there would not be enough time for a substitution to become fixed in the first place or they are looking at entire mtDNA studies where selection is affecting rates. This actually proves they are the ones cherry picking data to help support their views, when time and time again the scientists themselves *(Niel Howell)* say things like; "*There is* ***no evidence that any one factor explains this discrepancy****, and the possible roles of mutational hotspots (rate heterogeneity),* ***selection****, and random genetic drift and the limitations of phylogenetic approaches to deal with high levels of homoplasy are discussed.*"

> *"Sigurdardottir et al. (2000)* ***conclude that selection is unlikely to be a major factor that underlies the difference between phylogenetic and pedigree divergence rates****,...*" Howell et al 2003.
>
> "***As in other studies****, our estimation is* ***much higher than those obtained by phylogenetic methods****.*" - Santos 2005
>
> "***Whenever you see a time estimate in the evolutionary literature, demand uncertainty****."* - Dan Graur and William Martin 2004.

Such examples the critics point to are the rate studies, anomalies, in an attempt to undermine the consensus results. Such as Soodyall et al 1997 and Sigurðardóttir 2000, so we will look at each of these and explain why they are anomalies and why it is rather the critics that ignore the bigger picture and cherry pick random studies in desperation and run to these rare cases.

Soodyall et al 1997 study: *The founding mitochondrial DNA lineages of Tristan da Cunha Islanders.* Their aim was mostly population founder-effect / ancestry / lineage tracing, not primarily a mutation/substitution rate estimation *(although the mutation transparency came as part of the pedigree check).* The goal of the paper was actually: to understand founder lineages, to reconstruct population history and to determine how many female founders created the TDC maternal population Limitations: Small number of transmissions, limited statistical precision. Bottleneck founder isolated population with very limited diversity, typically constrains mutation detection/segregation. They also did not count heteroplasmies, and since they can turn into fixed substitutions if one variant eventually reaches 100% frequency, then obviously counting them and accounting for them is important to obtain an accurate substitution rate. But as stated earlier**, this was not their goal.** That is why they only counted homoplasmic substitutions *that were 100% fixed*. Why? Heteroplasmies drift up or down randomly, may disappear, may fix at the opposite base, may be somatic only and often last only 1 generation. Therefore for tracing haplotype ancestry back you only want fixed substitutions. Their small sample size, only 108 total transmissions between 75 related people would also be a problem for trying to obtain an accurate substitution rate. This is why Connell 2022 who tested essentially the same concept of an isolated island population and found many more substitutions based on the fact that there were more samples, **deeper lineages** and less filtering; *345 transmissions across 225 individuals. Found 9 mutations found (2 fixed + 7 heteroplasmic), and obtained* ***1 per 38 generations****.* Soodyall in contrast was **not scanning for every new mutation** in mother–child pairs the way Parsons, Howell, Connell and Helgason etc... did.

So:

- No special depth.
- No screening for low-level variants.
- No heteroplasmy analysis.
- No genealogical tree-based mutation-rate estimator.

This is why they did not calculate mutation rates, they did not estimate divergence times, they did not assume any generational interval and why you cannot find any generation time ages, they did not convert mutations into years. They simply identified the maternal founding haplotypes of the island. Not only that, the deepest and **only single** pedigree with limited transmissions (108) of shallow depth. So of course you wouldn't expect to see any new substitutions to arise and drift to fixation this rapidly, especially not looking for them. It really is this simple; They were only looking at ancestry results, not a substitution rate. It's not appropriate to use Soodyall (1997) to infer an accurate substitution rate and even they admit that this study was not for that. They knew the results were an anomaly, look at what they say and focus on the end where they place the substitution rate next to Parsons regardless of their results. "*We did not detect any mutations from 698 base pairs of sequence data from 75 individuals, which together accounted for 108 independent transmissions of mtDNA from mother to offspring.* ***Based on this observation, we estimate that the mtDNA mutation rate is no more than one new mutation every 36 transmissions."*** Parsons published one mutation every 33 transmissions, basically the same. The published rate from this anomaly of a study changes nothing for the overall substitution rate throughout the literature. So the "0 mutations" in this Tristan da Cunha study is not in conflict with the consensus = fast rates, it's just not a mutation rate study. Soodyall et al, was actually never supposed to be a substitution rate study or used as evidence for one, but rather a phylogeographic + demographic study. So using the study as an accurate substitution rate clock to attack all the others is the true definition of cherry picking. So it's typical that critics point to studies like this because they either do not know any better or they do and are disingenuous.

Another study the critics look to is Sigrún Sigurðardóttir 2000, an Icelandic population test of 272 **related** individuals in just 26 total pedigrees were studied, like Soodyall the Icelandic pedigrees derive from a relatively homogeneous founder population. This also can reduce ascertainment of parallel new mutations. But the real problem is, they catalogued but ignored heteroplasmic mutations. They literally only counted what had drifted to 100% homoplasmy. Also, if the same dataset were sequenced today with high-depth next-generation sequencing, they would likely detect: 20–50× more mutations, yielding a pedigree rate similar to Parsons 1997 *(1 per 33 generations)*. How do we know? Multiple studies that did this same thing, obtained those results. So yes, they were only counting the final endpoint of a mutation that becomes fixed, not the actual substitute rate across each generation. They admit this; "*In assessing the mutation rate from our study,* ***we have not used the information from heteroplasmic individuals****."* They explicitly did **not** include heteroplasmic sites in the main mutation-rate estimate. This exposes they chose to exclude all observed heteroplasmies to slow the rate down to move the rate slower to evolutionary phylogenetic assumptions rates. Not off to a good start are we? We can read this in their conclusion: "*For several reasons, it is* ***not clear*** *how to treat instances of heteroplasmy in the estimation of mutation rates.* ***Unlike authors of some studies, we have not included such instances in our estimate****."* They, like Soodyall, only ever counted fixed substitutions. This is why they published a rate of 3/705 = 0.0043 per generation, yet, observed a faster rate of **3/285 = .011** *(95% CI .0022 – .030).*

Since they had such stringent filtering of any questionable variants including what they thought might be somatic mutations, they were **all** removed in the final rate since they were not fixed. The fact is, they were actually removing and not counting true mutations. How do I know this? Neil Howell in 2003 published this; "*Sigurdardóttir et al. 2000 also raise the concern that many heteroplasmic mutations are somatic variants rather than true germline mutations.* ***However, their data clearly show that the heteroplasmic mutations in their lineages were inherited through multiple generations and therefore that they cannot be somatic.*** *The requirement for multigeneration transmission was a specific inclusion*

criteria for our analyses, and a survey of the other published studies indicates that inflation of the estimated k_{ped} *(table 2) by* ***somatic variants*** *is* ***untenable****. Even in the study reported by Parsons et al., which analyzed small pedigrees, the newly arising mutations* ***were detected in multiple family members and thus cannot be somatic.****"*

So yes, *Sigurdardóttir* is a flawed study to use for a true substitution rate for many reasons. So again, pointing to it to undermine the results of all the other studies is a clear case of cherry picking. I believe the problem is a combination of things such as the discrepancy whereSigurðardóttir et al. excluded many true mutations in the course of removing what they thought was somatic noise and also did not include heteroplasmic mutations in their final results including ignored all heteroplasmy below 30% frequency. Sigurdardóttir also used an extremely high confidence interval *(CI)* assuming Poisson counting of rare events. What are the consequences of having such a high and low CI? Well, their rate of 1/0.013 x 20 rate places the MRCA anywhere between 1,538 years ago and 22,727 years ago using the slowest rate (1/0.00088 x 20). Next there is statistical luck: small-*n* stochasticity. "*Small-n*" means they didn't see very many events *("n" = number of observations).* "Stochasticity" means randomness. In simple terms, when you're working with small numbers, random chance has a huge influence on your results. Basically they only caught 3 mutations. If just *one more* had appeared *(4 instead of 3)*, their mutation rate would jump by 33%. If there had been 6 instead of 3, it would *double*. That's how unstable a result can be when the dataset is tiny like this study was. This is like trying to guess how often it rains in Iceland after watching the weather for only one week. The true rate is much faster and consistent, this is why Connell et al who also tested a similar isolated island population also of icelandic populations with the same pedigree depth degree as *Sigurdardóttir* found the **exact opposite results** with many more mutations in contrast. *Sigurdardóttir* also noted that they moved the typical generation time in pedigree studies up from 20 all the way to 30. Not a real big deal but this means their rate also appears lower because their generational span was longer. These are just some of the reasons why they obtained such an anomaly and older MRCA, with a rate of 3/704 or 1 every 235 generations. Therefore this pedigree is just one in a very few studies that obtained inconsistent results and should not even count towards a true accurate substitution rate. This is why they still noted in their conclusions: The final statement in their study is that they admit pedigree studies are still the best and true clocks of history; *"If the aim is to estimate the average mutation rate either across the entire CR or across one or both of its hypervariable regions,* ***then estimates obtained by directly counting the mutations in pedigrees are unbiased for this rate****, regardless of the degree of rate heterogeneity, unless, as seems unlikely, some sites mutate so quickly that pedigree studies will* ***fail to observe two mutations at the same site*** *in distinct generations within the pedigree."* This is basically saying that the rates we observe in pedigree studies are spot on, unless mutations are flipping over in the same place *(multiple hit)* that throws off the rate and makes it slower. So now imagine if a mutation arises in 1 of the 19 hot spots in HVR1 every few generations, but the next mutation does not occur in one of the other 18 spots but flips the same spot again. The mutation rate would essentially be much slower as it would not be seen or counted as since it went undetected. This would actually slow the mutation rate down, and they admitted that this could have been a possibility as well in this study. This is more validation that these rapid rates cannot even remotely be slowed down to reach evolutionary rates because not only is selection not having an effect in these regions, but mutations may even be flipping over in the same place and going uncounted which actually makes the true rate even **faster!** The news just keeps getting worse for evolution with new discoveries like this. This process can reduce the measured rate by another factor of 10. So in conclusion of this study; They did not count heteroplasmic mutations *(even if detected)*. They removed all mutations they deemed somatic, which Howell later confirmed were actually true mutations and they only counted mutations that had already become fixed prior. This was far from an accurate substitution rate study, perhaps why the critics like to point to it. They tend to love outliers and ignore the trend. Regardless, in the

study they admitted something vital, and that is that pedigree mutation rates in this region are unbiased since they are neutral and that the only problem one can have is if mutations hit the same spot twice which can only speed slow the rate down since they may not notice the mutation. Meaning the rate is still fast, and nothing can slow it down except unseen mutations occurring in the same spot occurring going un-noticed.

Anytime you read a study and they omit heteroplasmy, you can be sure that this single limitation hides the majority of mtDNA mutations and slows the rate down dramatically, because most new germline mtDNA mutations start at 1–5% frequency and what is lost are mostly lost within the first generation anyway. Parsons noted that;

- 70–80% of heteroplasmies disappear in the first generation (not transmitted, or transmitted at <1% and effectively gone).
- 15–25% transmit to the next generation as heteroplasmic (still present, but frequency may shift up or down).
- 5–10% eventually become fixed substitutions (can be a few generations later; extremely random).

Howell et al (2003) confirmed: Out of 45 mother–offspring heteroplasmies: 80% changed frequency significantly. Most that persisted were around the 10–40% range and fixation events tended to begin around 60–90%. So ignoring and not counting heteroplasmy at all, or at different frequencies, will always undermine a true accurate mutation rate.

While some of the studies I covered earlier may have published a slower rate, somewhere in their study they admitted what the true rate was and then filtered or calibrated it after. We see this time and time again and one example of that was Heyer et al 2001. A deep rooted pedigree study where they stated that if they ignore mutational hot spots the rates align more with evolutionary phylogeny, but when they include these regions, they confirm typical pedigree rates. They admitted their study by stating: "*Confirming earlier findings of much greater mutation rates in families than those based on phylogenetic comparisons.*" The theme continues as usual.

Animal Species

You would think that if evolution was true, clearly one animal species on earth would show a deep ancestry in their genome. Especially since the regions being tested are as near neutral as you can get. Instead we always find low genetic diversity and rapid rates of change. These two things cannot exist at the same time and for evolution to be true. The genetic data is so in favor of the Biblical creationist timeline that the critics have to resort to looking at rare anomaly studies or studies that calibrate/anchor to known ages. The reality is, nothing alive shows deep ancestry and we are going to be looking at these same regions in diverse species to expose this reality.

Therefore another line of refutation of the critics and these mutation rates is to broaden the scope beyond humans and look at diverse animal species worldwide. Multiple animals have been tested using the same pedigree-based methods, and all that have ever been tested using these methods have validated the same thing, a very recent MRCA and far from any evolutionary fossil record date. The theme is consistent and falsifies evolution time and time again.

Whales

Whales show that phylogenetic estimates fail and pedigree rates land on known historical events. This study titled "*Wild pedigrees inform mutation rates and historic abundance in baleen whales*" by Marcos Suarez-Mendez et al who tested the control region. The final estimate of μ (mutation rate) for the control region was 4.3 x 10^{-6} Per-site, per-year or 1 substitution every 19–52 generations (*IQR: 1.55 × 10−6 to 8.85 × 10−6 =IQR rates: ~9–144 generations per substitution*). The single mutation rate estimate from baleen whales [bowhead whale] was 7 × 10^{-6} to 1.05 × 10^{-5} this works out to be 1 substitution every 8–32 generations or 1 substitution occurs about every 32–88 years, depending on whether their generation time is closer to 4 or 11 years. Results matched known historical records for a bottleneck from commercial whaling and yet again refuted phylogenetic fossil rates.

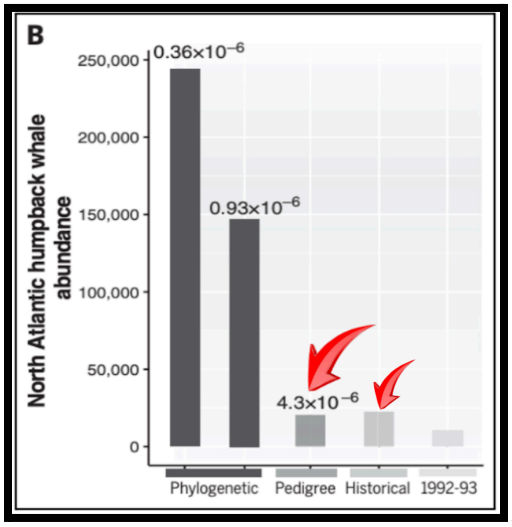
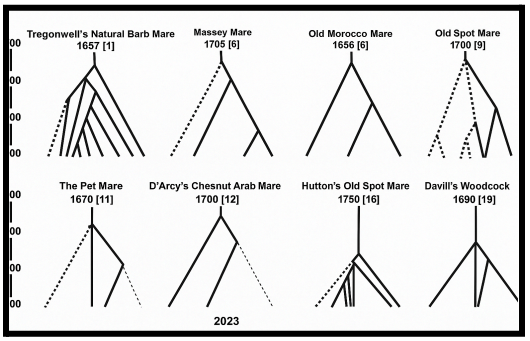


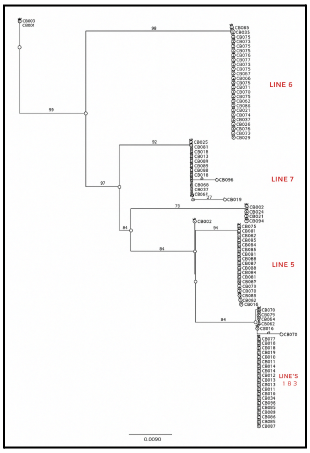
***Figure 7**. Red arrows show known historical whaling events match the pedigree mutation rate and not the evolutionary assumption phylogenetic rates seen on the left side of the chart.*

Horses (Equus ferus caballus)

Hill *et al. 2002, "History and integrity of Thoroughbred dam lines revealed in equine mtDNA variation"*. Studied thoroughbred racehorses — historically recorded foundation mare families from the General Stud Book (England). 100 Thoroughbreds from 19 prominent maternal families were tested. When they tested these Thoroughbred horses, they found that nearly half of the known female bloodlines didn't match what the official pedigree records said. Many mares that were thought to share the same female ancestor actually didn't, based on their mitochondrial DNA. After comparing these mismatches to the old breeding records, the researchers concluded that most of the mix-ups happened a long time ago, when the breed was first being organized, likely due to mares being mislabeled, swapped, or confused in the early records. A few of the errors may have happened later, between the 1800s and 1980. **By combining the DNA evidence with historical documents, the researchers were able to identify which original mares were the true founders of certain lines, some going as far back as the 1657 or 34 to 43 generations of matrilineal descent.** They gained new insights into how the Thoroughbred breed actually began and developed all based on the pedigree mutation rate accuracy.

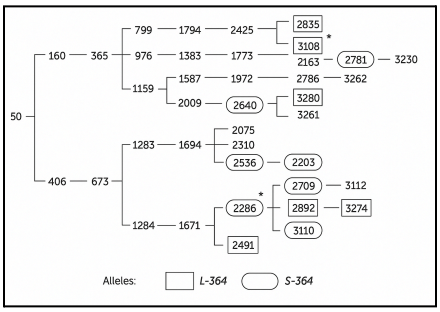


The Cleveland Bay horse study by Dell et al confirmed the accuracy yet again of these pedigree studies by confirming four female founder lines identified from the mtDNA haplotypes correspond to documented mares that lived during the breed's formation in northern England in the 17th–18th centuries, when the Cleveland Bay developed from crosses of local packhorses with imported hot-blooded stock. The founding mares for these lines lived roughly between 1650 – 1750 AD, during the same era as the early Thoroughbred founders. This aligns with historical accounts that the Cleveland Bay arose from local packhorses crossed with imported "hot-blooded" stallions *(Arabian, Barb, or Turkoman types).* **The molecular data precisely mirrored the recorded pedigrees**, confirming that: The deepest-rooted Cleveland Bay pedigree in Dell et al. *(2020)* is Line 1 & 3 *(Clade A1 / Haplotype CBHap1)*, descending from mares living in 17th-century northern England, representing the oldest surviving maternal lineage in the breed and aligning perfectly with both historical and pedigree evidence. The evolutionary phylogenetic "clock" rates drastically overestimate time depth for such recent lineages and fails to land on any appropriate date.



Holstein cattle (Bos taurus)

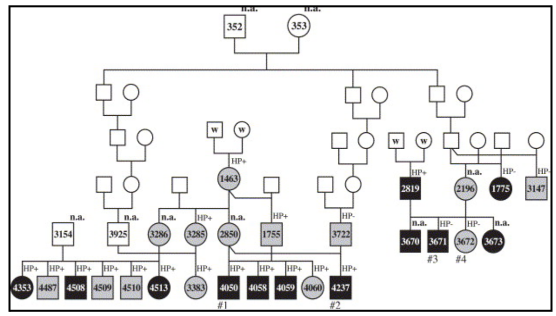
Domestic cattle provide another example confirming this pattern. In a pedigree study, Koehler et al. analyzed 174 Holstein individuals representing 35 maternal lineages, focusing on variation in the mitochondrial control region (D-loop). A C/T substitution at position 364 was found to differ between mothers and daughters in 13 lineages, and additional variation at nucleotide 5602 was observed in one lineage within only three generations. Because the Holstein breed was standardized during the 18th–19th centuries in the Netherlands and Germany, the effective most recent common ancestor for these haplotypes almost certainly lived within the past 200–300 years, consistent with herd-book history rather than the tens of thousands of years inferred from phylogenetic rate estimates. This data indicated that all Holstein mitochondrial lineages trace back to a single maternal ancestor from the period of breed formation, providing a strong empirical calibration of observed pedigree mutation rates. The study also demonstrated that mtDNA haplotypes can be replaced between generations due to genetic drift during the mitochondrial bottleneck.



Cheetah (Acinonyx jubatus)

Another striking piece of the puzzle comes from cheetah pedigree-based mtDNA studies. Burger et al. (2004) sequenced the full mitochondrial control region (D-loop) and ND5 gene in African and Iranian cheetahs, revealing only three haplotypes separated by just 2–4 substitutions, a vivid demonstration of how extraordinarily low modern cheetah maternal diversity really is. The maximum difference observed in their dataset was 4 substitutions across 1,218 bp, a genetic distance of roughly 0.0033 under the standard two-lineage coalescent model. When this divergence is paired with a typical pedigree-based mutation rate of $\mu = 10^{-5}$ per site per generation, the maternal coalescent time collapses dramatically from the evolutionary timeframe. The resulting estimate places the mitochondrial MRCA for these sampled cheetahs at 160 generations, or 1,000 years *(assuming 6 years per generation).* Charruau et al. (2011) expanded the picture, identifying nine fixed mtDNA haplotypes across the species. These haplotypes differ by nine diagnostic substitutions, which—under the same coalescent model and the same pedigree-calibrated rate ($\mu = 10^{-5}$)—yield an MRCA of roughly 370

generations, or 2,200 years. In other words, even when considering all known maternal lineages, the mtDNA data consistently point to a remarkably recent mitochondrial ancestor within the last 1–2.2 thousand years. This stands in stark contrast to the widely cited evolutionary bottlenecks at 100,000 years and 10,000–12,000 years ago. Once again, the genetic math refuses to align with the traditional narrative. Taken together, these findings are consistent with predictions made by Biblical creationists. Since science ultimately evaluates hypotheses using statistical probability (P-values), the consistent results observed across diverse species lend strong empirical support to this model. The rapid mitochondrial mutation rates shared in this study in humans, whales, horses, cattle, and cheetahs, including many other documented organisms make the observed pattern highly unlikely to be coincidental.



Domestic Cats

The genetic clock derived from the mummified cats did not merely *approximate* the timeline of Egyptian history, it landed on it with astonishing precision. This is an example of how we can go back thousands of years of using DNA differences to reconstruct a mutation rate that validates the Biblical time. Kurushima and her team predicted that the maternal ancestor of these cats mummified around 664 - 332 BC, lived between 1.97 to 7.5 thousand years before their mummification, placing their origins between 7,600 and 2,600 B.C.. That upper boundary, 2.6 kya before Christ, aligns perfectly, within with the exact era when the first Egyptian artisans carved the hieroglyph for cats "miu" (𓃠). This wasn't a broad guess spanning a few generations, it was a deep genealogical timestamp so exact it could be read alongside hieroglyphs in a museum. In this amazing study titled: *Cats of the Pharaohs: Genetic Comparison of Egyptian Cat Mummies to their Feline Contemporaries* by Jennifer D Kurushima et al. The team sequenced the mitochondrial control region *(CR)* of mummified Egyptian cats that were mummified between *(664–332 B.C.).* They sequenced 246 and 402 base pairs per mummy, using three overlapping amplicons. When complete, the fragment spans positions 16814–206 of the feline CR *(the "Sylvester Reference Sequence"* These regions are the last part of HVR-I *(near the 16.8 kb end of the reference),* the central conserved domain *(the non-hypervariable block around the D-loop/CR origin)* and the first 200 bp of HVR-II *(up to position 206)*. They mapped each mummy's CR haplotype *(mitotype*) onto a global cat mitotype dataset and then computed divergence times *"as in López et al. (1997)",* bracketing the dates with "*neutral*" and "*fast*" derived from other mitochondrial genes *(12S and ND2)* to give a range. All three mummy mitotypes: G, C, and the B/D/J cluster, belong to the same broad Near Eastern / Egyptian clade identified by Grahn et al. *(2011)*, distinct from the Western European mitotype A group. That's why the authors concluded: *"All three mummies are representative of closely related mitotypes that are still present in the modern Egyptian population... indicating that modern cats of Egypt are descendants of local ancient populations."* Using this data, they estimated that the mummies' mitotypes originated around 1.97 to 7.5 kya before the cats lived. The study doesn't measure a new mutation rate, but it shows that the diversity of modern Egyptian cat mtDNA goes back only a few millennia, not tens of thousands of years. Across the 402-bp control region, the maximum divergence within the mummy-clade is only about 5–6 substitutions, with most mitotypes separated by just 2–4 differences. With a mutation rate common in the D-Loop; $\mu \approx 3\times10^{-6}$ to 1.1×10^{-5} per site per generation *(with 95% CIs spanning ~10^{-7} to 2×10^{-5} depending on D and g)*. This rate, demonstrated in direct parent-offspring sequencing studies, yields a 2,000-year maternal MRCA for the mummified cats. In other words, the genetics land exactly where the archaeology and art history already point: the emergence of domestic cats in Egypt during the very centuries they first appear in inscriptions, iconography, and recorded cultural life. The earliest hieroglyph representing a cat, written as miu or miut (𓃠) appears during the

Old Kingdom, around ca. 2,600 B.C. *(Fourth Dynasty)*. The word *miu* literally imitates the cat's meow *("the one that mews")*. The symbol depicts a seated cat with its tail curled forward. Another of the earliest examples comes from Saqqara tomb inscriptions and administrative lists referencing animals and household pets and is dated 2,400–2,150 BC. Now to compare:

Images; roughly 2,400–2,150 B.C.

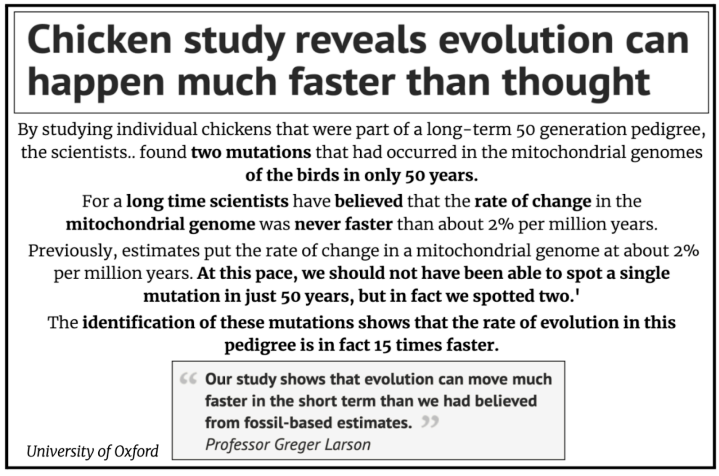
The cat ancestor window starts at; 2302 B.C.

This is an overlap of 51 - 98 years.

That level of alignment between a molecular coalescence date, the mutation rate combined with the archaeological record is unheard of in ancient DNA evolutionary phylogenetic studies, where uncertainty often spans millennia. For the first time, genetics, archaeology, and ancient writing all come together to tell one story in unison: that by the dawn of Egypt's Old Kingdom, the wild hunter of the marsh had already crossed the threshold, from wilderness to worship. The DNA of the mummified cats does not merely trace ancestry; it confirms a moment when the wild became tame, and history purred its approval. This corroborating evidence confirms yet again that the observed molecular clocks we creationists point to for confirmation of "Eve" living recently and a global bottleneck in the recent history are the true accurate clocks of history and tell the story perfectly while the evolutionary assumption rates derived from fossils are simply retrofitting the data to fit a failed paradigm. This amazing study again shows that the HVR regions in the CR make the best clocks because of their neutrality ticking mutations. Results consistently land on known historical documented ages even when going back thousands of years like in this case. This again validates the Biblical timeline and refutes the vast ages presented in evolutionary literature and falsifies the fossil record time and time gain. The genes said; *"this is when the modern day domestic cat became ours,"* and Egypt's own monuments nodded in agreement.

Chickens (Gallus Gallus)

A remarkable opportunity to connect experimental mutation rates with real-world population history comes from combining the deep-rooted 50-generation chicken pedigree of Alexander et al. (2015) combined with the phylogeographic dataset of Yonezawa et al. The Alexander pedigree, spanning 385 documented mitochondrial transmissions, produced one of the most precise vertebrate mtDNA rate estimates to date, an astonishing fast rate of 3.13×10^{-7} mutations/site/year. They even admit in the news article titled: Chicken study reveals evolution can happen much faster than thought, that*"This is **substantially higher than avian rate estimates based upon fossil calibrations**."* Since this was a deep rooted pedigree, most selection and heteroplasmy drift already took effect, this is vital in this kind of study since we are dealing with the entire mtDNA and its mutation rate. With this empirically grounded rate in hand, we can turn to the Malagasy chicken D-loop dataset of Yonezawa et al., which provides the missing half of the equation to obtain a MRCA: observed genetic diversity, haplogroup frequencies, pairwise distances (π), and phylogenetic branching patterns. Plugging



Chicken study reveals evolution can happen much faster than thought

By studying individual chickens that were part of a long-term 50 generation pedigree, the scientists.. found **two mutations** that had occurred in the mitochondrial genomes **of the birds in only 50 years.**

For a **long time scientists** have **believed** that the **rate of change** in the **mitochondrial genome** was **never faster** than about 2% per million years.

Previously, estimates put the rate of change in a mitochondrial genome at about 2% per million years. **At this pace, we should not have been able to spot a single mutation in just 50 years, but in fact we spotted two.'**

The **identification of these mutations shows that the rate of evolution in this pedigree is in fact 15 times faster.**

"Our study shows that evolution can move much faster in the short term than we had believed from fossil-based estimates."
Professor Greger Larson

University of Oxford

these real population distances into the standard two-lineage coalescent model (T = d / 2μ) allows us to directly calculate the mitochondrial MRCA ages for each population group. The results are astonishingly recent. For all Malagasy chickens, the reported π = 0.00141 yields an MRCA of only 2,300 years. For East African chickens, π = 0.00096 gives an even younger MRCA of 1,500 years. The Malagasy population is further structured into Highland and Lowland lineages, but because π values were not reported separately, their MRCA estimates fall within the same expected window *(roughly 1,500–3,000 years)* given the narrow global chicken mtDNA diversity. When expanding beyond Madagascar to the entire Indian-Ocean / West Asian / African C2 lineage, typical chicken D-loop diversities ($\pi \approx 0.0010–0.0020$) correspond to MRCA ages of only 1,600–3,200 years. In other words, when a directly measured, high-resolution pedigree rate is applied to a real-world, historically structured population, the mitochondrial clock repeatedly collapses long evolutionary timelines into a surprisingly tight, recent, and highly consistent range across all populations examined. Rather than stretching back even just tens of thousands of years, the true pedigree-based mitochondrial ancestry of global chickens converges precisely within the last two to three millennia, and the data, from tightly controlled pedigrees to diverse island-wide populations, align with striking coherence. The first sentence of the news article covering this study states; *"A new study of chickens overturns the popular assumption that evolution is only visible over long time scales."* Every time they observe a pedigree mutation rate inside a species they end up shocked. In this case, expecting a slower mutation rate because of how far back and deep rooted the pedigree was. They were not expecting substitutions to arise that fast and they were expecting selection to have removed these mutations over successive generations, allowing the clock to slow down and match evolutionary time. Yet they did not, the opposite was found. These consistent themes are ignored by the critics who love to ignore and latch onto the few favorable studies that confirm their story.

DISCUSSON

The consistent theme of rapid mutation rates across diverse species both land and aquatic including the plant kingdoms that just so happens to fall directly in line with the Biblical timeline cannot be just by chance and should not be ignored. These rates refute the fossil record and the evolutionary timeline every time unless they are forced to fit into the process. This tension is precisely what science journalist Ann Gibbons (1998) was referring to in her Science report: *"Evolutionists are most concerned about the effect of a faster mutation rate."* They should be, this means they cannot accurately explain the diversity of life on this planet and fail to make any accurate testable predictions. Meanwhile the creationists are making the predictions and getting them correct based on the biblical timeline.

An interesting thing to consider is *t*he pedigree mutation rate studies in humans also align perfectly with genealogical rates *(Douglas L. T. Rode et al 2004)* who used multiple mathematical models to test the date for a MRCA and location. They quote: *"In particular, the* ***MRCA of all present-day humans lived just a few thousand years ago in these models****."* They also noted that: *"the simulations produce a mean MRCA date of 1,415 BC and a mean IA* (Identical Ancestors Point) ***date of 5,353 BC* (7,378 years ago). *Interestingly, the MRCAs are nearly always found in eastern Asia****"*. Notice this is not Africa but rather outside Africa to the east in Asia towards the direction of Babel? Coincidence? I think not. Then we have unbroken chains of genealogies recorded in ancient texts tracing royalty all the way back to Noah (*The Lambeth Roll* or *The Canterbury Roll* depending on the copy). Some of the mutation rates even confirm some of the post flood material lineage ancestors of particular ancestry matching the expansion time after the flood and from Babel via the arrival of harmful SNV's *(Jacob A. Tennessen et al 2012)* who published their results stating; *"accelerated population growth began 5,115 years ago with a per generation growth rate of 1.95% and 1.66% for EAs and AAs, respectively"*. All of this including

matching known population growth rates and starting from just 8 people, the origin of math, astrology, medicine, metallurgy, brewing of beer, irrigation, leather tanning, construction of megalithic structures, written history and the arrival of cultures and civilizations all start around the same time, including the fact that the oldest deltas, trees, deserts, coral cays *(islands made of coral and sand)*, helium and argon diffusion rates, all go back to the same timeframe and the oldest living organisms that do not die from old age like deep-water black sea corals all converge on this same date as well. The massive amount of parsimony from vastly different fields that all match pedigree mutation rates more than confirm their accuracy and precludes evolution theory from being even remotely a reliable theory. The simplest story remains the truest: we all descend from a single, recent origin and exploded in population growth after a global bottleneck in the recent past including animals. We did not split from a primate ancestor millions of years ago evolving over thousands of years to modern man nor are related to fish, banana plants and grass. Genetic similarity does not mean genetic relation, these two things are conflated to make evolution seem true. Rather, all of the evidence above does not converge by chance, it is obvious to anyone who is unbiased and logical that observed mutation rates and population histories the convergence of genealogical, genetic, and historical data points unmistakably to a recent, unified human origin. The empirical coherence and observable data favor design and descent from a single founding population in the recent past, not deep evolutionary time.

Without an evolutionary deep time mindset and calibrations made to some of these molecular clocks, you cannot even remotely get evolutionary dates. The observed rates confirm the Biblical timeline and only when scientists adjust and calibrate the data to the evolutionary timeline can they force it to give results in favor of deep time. Genetic data refutes the fossil record and the dates that come with them, confirming the title of this study: When Facts Falsify Fossils.

The goal of evolution theory is to explain the diversity of life on earth, and mutations are key to that process. Mutations are what causes diversity to go up and if mutation rates are fast and they require them to be slow, then evolution cannot accurately explain the diversity of life. This is why evolution has failed to make any accurate testable predictions using their phylogenetic mutation rate, rather they have to adjust the rate to match the radiometric dates. It really is just the paradigm driving the conclusions. They say, it must be old therefore the observable clocks must all be wrong and we need to change them. They never think for even a minute that maybe the alternative is wrong.

Take this example, they say *"Dinosaurs are old because we have radiometrically dated them to 65 + million years ago"*. Actually what they have done is test the rocks around them, a big difference. When we actually test the fossils themselves they scream recently. They have detectable levels of uncontaminated carbon 14 in them and we see the same C14 in coal, oil and supposedly ancient petrified wood. They also have multiple forms of soft tissue still preserved in them. We see ancient rock art with directions of dinosaurs and therapsids. We have documented stories about seeing dinosaurs, we have legends of dragons from around the world. So we have all this evidence directly refuting what the rocks are saying, and they want us to ignore all of that in favor of a dating method that is only as accurate as a coin toss. See my paper titled: The Illusion of Deep Time: Systematic Discordant Radiometric Ages and the Myth of an Ancient Ocean Floor. What you have learned today is validation that phylogenetic evolutionary rates are not the best explanation and frankly not even a good one, but rather the Biblical model of ancestry is. The accurate predictions will continue to flow and continue to refute evolution theory and the critics will continue to claim we are wrong because the majority disagrees while ignoring the observable empirical data that has never been falsified. Take your pick: are you on the side of science or are you going to let emotion and personal bias decide what you want to be true?

RESOURCES

Heyer, E., Zietkiewicz, E., Rochowski, A., Yotova, V., Puymirat, J., & Labuda, D. (2001). Phylogenetic and familial estimates of mitochondrial substitution rates: Study of control-region mutations in deep-rooting pedigrees. *American Journal of Human Genetics, 69*(5), 1113–1126. doi: 10.1086/324024

Howell, N., Kubacka, I., & Mackey, D. A. (1996). How rapidly does the human mitochondrial genome evolve? *American Journal of Human Genetics, 59*(3), 501–509. https://pmc.ncbi.nlm.nih.gov/articles/PMC1914922/

Parsons, T. J., Muniec, D. E., 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

Nature Genetics 18(3), 1998. By Ann Gibbons

Sigurðardóttir, S., Helgason, A., Gulcher, J. R., Stefánsson, K., & Donnelly, P. (2000). The mutation rate in the human mtDNA control region. *American Journal of Human Genetics, 66*(5), 1599–1609. https://www.sciencedirect.com/science/article/pii/S0002929707629905

Sam, K.-K., Chia, O. K. S., & Chong, V. C. (2021). Complete mitochondrial genomes of *Paedocypris micromegethes* and *Paedocypris carbunculus* reveal conserved gene order and phylogenetic relationships of miniaturized cyprinids. *Frontiers in Ecology and Evolution, 9*, 662501. https://doi.org/10.3389/fevo.2021.662501

Howell, N., Elson, J. L., Turnbull, D. M., & Herrnstadt, C. (2007). Relative rates of evolution in the coding and control regions of African mtDNAs. *Molecular Biology and Evolution, 24*(10), 2213–2221. https://doi.org/10.1093/molbev/msm152

Árnadóttir, E. R., Moore, K. H. S., Guðmundsdóttir, V. B., Ebenesersdóttir, S. S., Guity, K., Jónsson, H., Stefánsson, K., & Helgason, A. (2024). The rate and nature of mitochondrial DNA mutations in human pedigrees. *Cell, 187*(15), 3904–3918.e8. https://doi.org/10.1016/j.cell.2024.05.022

Charruau, P., Fernandes, C., Orozco-terWengel, P., Peters, J., Hunter, L., Ziaie, H., Jourabchian, A., Jowkar, H., Schaller, G., Ostrowski, S., Vercammen, P., Grange, T., Schlötterer, C., Kotze, A., Geigl, E.-M., Walzer, C., Bürkle, M., & Burger, P. A. (2011). *Phylogeography, genetic structure and population divergence time of cheetahs in Africa and Asia.* **Molecular Ecology, 20**(4), 706–724. https://doi.org/10.1111/j.1365-294X.2010.04986.x

Dell, A. C., Curry, M. C., Yarnell, K. M., Starbuck, G. R., & Wilson, P. B. (2020). *Mitochondrial D-loop sequence variation and maternal lineage in the endangered Cleveland Bay horse.* PLoS ONE, 15(12), e0243247. https://doi.org/10.1371/journal.pone.0243247

Hill, E. W., Bradley, D. G., Al-Barody, M., Ertugrul, O., Splan, R. K., Zakharov, I., & Cunningham, E. P. (2002). *History and integrity of Thoroughbred dam lines revealed in equine mtDNA variation. Animal Genetics, 33*(4), 287–294. https://doi.org/10.1046/j.1365-2052.2002.00870.x

Koehler, C. M. (1991). Replacement of bovine mitochondrial DNA by a sequence variant within one generation. *Genetics, 129*(1), 247–255. https://doi.org/10.1093/genetics/129.1.247

Morelli, L. (2014). Mitochondrial DNA lineages of Italian Giara and Sarcidano horses. *Genetics and Molecular Research, 13*(4), 8241–8257. https://doi.org/10.4238/2014.October.20.1

Burger, P. A. (2004). Analysis of the mitochondrial genome of cheetahs (*Acinonyx jubatus*) with neurodegenerative disease. *Gene, 338*(1), 111–119. https://doi.org/10.1016/j.gene.2004.05.020

Luis, C. (2002). Variation in the mitochondrial control region sequence between the two maternal lines of the Sorraia horse breed. *Genetics and Molecular Biology, 25*(3), Article e00010. https://doi.org/10.1590/S1415-47572002000300010

Bowling, A. T., Del Valle, A., & Bowling, M. (2002). A pedigree-based study of mitochondrial D-loop DNA sequence variation among Arabian horses. *Animal Genetics, 33*(2), 97–103. https://doi.org/10.1046/j.1365-2052.2000.00558.x

Soodyall, H., Jenkins, T., Mukherjee, A., Du Toit, E., Roberts, D. F., & Stoneking, M. (1998). The founding mitochondrial DNA lineages of Tristan da Cunha islanders. *American Journal of Physical Anthropology, 104*(2), 157–166. https://doi.org/10.1002/(SICI)1096-8644(199710)104:2<157::AID-AJPA2>3.0.CO;2-W

Santos, C., Montiel, R., Sierra, B., Bettencourt, C., Fernandez, E., Alvarez, L., Lima, M., Abade, A., & Aluja, M. P. (2005). Understanding differences between phylogenetic and pedigree-derived mtDNA mutation rate: A model using families from the Azores Islands (Portugal). *Molecular Biology and Evolution, 22*(1), 149–155. https://doi.org/10.1093/molbev/msh262

Madrigal, L., Meléndez-Obando, M., Villegas-Palma, R., Barrantes, R., Azofeifa, J., Ramírez, E., Saenz, G., & Stone, A. C. (2012). High mitochondrial mutation rates estimated from deep-rooting Costa Rican pedigrees. *American Journal of Physical Anthropology, 148*(3), 327–333. https://doi.org/10.1002/ajpa.22052

Howell, N., Howell, C., & Elson, J. L. (2003). Does selection explain the differences between phylogenetic and pedigree rates of mtDNA evolution? *Annals of Human Genetics, 67*(1), 1–14. https://doi.org/10.1046/j.1469-1809.2003.00009.x

Carter, R. W., Criswell, D., & Sanford, J. (2008). The "Eve" mitochondrial consensus sequence. *Journal of Creation, 22*(2), 84–92.*

Budowle, B., Allard, M. W., Wilson, M. R., Chakraborty, R., & Schanfield, M. S. (1999). Forensic and evolutionary implications of the analysis of mitochondrial DNA variation. *Annals of the New York Academy of Sciences, 882*(1), 197–207. https://doi.org/10.1111/j.1749-6632.1999.tb08575.x

Lundström, R., Tavaré, S., & Ward, R. H. (1992). *Estimating substitution rates from molecular data using the coalescent.* Proceedings of the National Academy of Sciences, 89(13), 5961–5965. https://doi.org/10.1073/pnas.89.13.5961

Thorne, J. L., Kishino, H., & Painter, I. S. (1998). Estimating the rate of evolution of the rate of molecular evolution. *Molecular Biology and Evolution, 15*(12), 1647–1657.*

Sanderson, M. J. (2002). Estimating absolute rates of molecular evolution and divergence times: A penalized likelihood approach. *Molecular Biology and Evolution, 19*(1), 101–109.*

Thorne, J. L., & Kishino, H. (2002). Divergence time and evolutionary rate estimation with multilocus data. *Systematic Biology, 51*(5), 689–702.*

Drummond, A. J., Ho, S. Y. W., Phillips, M. J., & Rambaut, A. (2006). Relaxed phylogenetics and dating with confidence. *PLoS Biology, 4*(5), e88. https://doi.org/10.1371/journal.pbio.0040088

Yang, Z. (2006). *Computational Molecular Evolution.* Oxford University Press.

Suárez-Méndez, M., et al. (2023). Wild pedigrees inform mutation rates and historic abundance in baleen whales. *[Journal name—likely Molecular Biology and Evolution or Molecular Ecology; confirm exact citation and*

year once available].

Zuckerkandl, E., & Pauling, L. (1962). Molecular disease, evolution, and genetic heterogeneity. In *Horizons in Biochemistry* (pp. 189–225). Academic Press.

Kimura, M. (1983). *The Neutral Theory of Molecular Evolution.* Cambridge University Press.

Sigurðardóttir, S., Helgason, A., Gulcher, J. R., Stefánsson, 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/10.1086/302902

The Metropolitan Museum of Art. (n.d.). *Relief with hieroglyphic label "Lord of Cats' Town" [Egyptian, Old Kingdom, ca. 2353–2150 B.C.]*. Retrieved November 11, 2025, from https://www.metmuseum.org/art/collection/search/551093

Kurushima, J. D., Grahn, R. A., & Lyons, L. A. (2012). *Cats of the Pharaohs: Genetic comparison of Egyptian cat mummies to their feline contemporaries. Journal of Archaeological Science, 39*(10), 3217–3223. https://doi.org/10.1016/j.jas.2012.04.030

Nailor, M. (with editorial contributions by Budinsky, D.). (2025). *The illusion of deep time: Systematic discordant radiometric ages and the myth of an ancient ocean floor. Truth In Research.* https://doi.org/10.5281/zenodo.16956858

Vaesen, K., Scherjon, F., Hemerik, L., & Verpoorte, A. (2019). *Inbreeding, Allee effects and stochasticity might be sufficient to account for Neanderthal extinction. PLOS ONE, 14*(11), e0225117. https://doi.org/10.1371/journal.pone.0225117

Mumm, S., Whyte, M. P., Thakker, R. V., Buetow, K. H., & Schlessinger, D. (1997). *mtDNA analysis shows common ancestry in two kindreds with X-linked recessive hypoparathyroidism and reveals a heteroplasmic silent mutation*. American Journal of Human Genetics, 60(1), 153–159. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1712538

Connell, J. R., Benton, M. C., Lea, R. A., Sutherland, H. G., Chaseling, J., Haupt, L. M., Wright, K. M., & Griffiths, L. R. (2022). *Pedigree derived mutation rate across the entire mitochondrial genome of the Norfolk Island population*. Scientific Reports, 12, Article 6827. https://www.nature.com/articles/s41598-022-10530-3

Cvijović, I. (2018). *The effect of strong purifying selection on genetic diversity*. Genetics, 209(4), 1235–1278. https://doi.org/10.1534/genetics.118.301058

Chicken study reveals evolution can happen much faster than thought | University of Oxford

B. Bonne-Tamir, M. Korostishevsky, A. J. Redd, Y. Pel-Or, M. E. Kaplan and M. F. Hammer, Maternal and Paternal Lineages of the Samaritan Isolate: Mutation Rates and Time to Most Recent Common Male Ancestor, Annals of Human Genetics, Volume 67 Issue 2 Page 153 – March 2003

Guo, M., Yuan, C., Tao, L., Cai, Y., & Zhang, W. (2022). Life barcoded by DNA barcodes. *Conservation Genetics Resources, 14*(4), 351–365. https://doi.org/10.1007/s12686-022-01291-2

Evolution and Functional Impact of Rare Coding Variation from Deep Sequencing of Human Exomes by Jacob A. Tennessen", Abigail w. Bigham, Timothy D. O'Connor, Wenqing Fu, Eimear E. Kenny', Simon Gravel, Sean. Science, 06 Je 2093. Issue 6090. op. 64-69

Todd Wood 2012 Ancient mtDNA Implies a Nonconstant Molecular Clock in the Human Holobaramin.

Published: 30 September 2004 Modelling the recent common ancestry of all living humans by Douglas L. T. Rode L, Steve Olson & Joseph T. Chang Nature 431, 562-566(2004)

News article: Sweeping gene survey reveals new facets of evolution by Marlowe Hood