

The Hidden Clock of Humanity

A pedigree-based coalescence and fixation analysis of the human mitochondrial control region

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ABSTRACT

We test whether the pedigree, or germline, substitution rate of the human mitochondrial control region is compatible with a recent origin of humanity. Pooling the deeper-rooted, HVR-compatible pedigree studies used in our principal high-rate subset; Moreau et al. (2009), Madrigal et al. (2012), Howell et al. (1996), and Santos et al. (2005), yields an exact unweighted harmonic mean of one substitution every 19.947 generations per lineage. We deliberately describe this as a high-end mean: it represents the faster portion of the observed pedigree-rates distribution, but it is neither the single fastest reported rate nor an average of the entire literature. Projected across the candidate biblical interval of approximately 225 to 380 generations, this pooled rate predicts roughly 22.6 to 38.1 pairwise substitutions between two descendant lineages. Both of the deepest empirical comparisons considered here fall within that range. The African American maximum of 32 differences lies comfortably inside the predicted interval, while the all-database maximum of 36 differences, the single deepest pair among the eight regional databases examined, also remains within the upper end of the pedigree-rate expectation. Thus, at the pooled high-end pedigree rate used in the principal analysis, neither the representative 32-difference comparison nor the maximum 36-difference comparison requires a coalescence age older than the upper chronological limit being tested. At the population-average level, an African American mean pairwise diversity of 14.1 is generated within 6,000 years by a rate of approximately one substitution every 42.6 generations (Árnadóttir et al. 2024). We set these genetic results beside further lines of evidence developed elsewhere in the study: the recent origin of most human protein-coding and deleterious variation and the maximum-likelihood onset of accelerated population growth (Tennessen et al. 2012; Fu et al. 2013), the arithmetic of population growth from a small post-Flood founding number, the recent genealogical convergence of all living humans (Rohde et al. 2004), the abrupt onset of documented history within the last 5,000 years, the internal chronology of the Biblical genealogies, and a decay-kinetics test in which the fragmentation of DNA from a non-frozen, temperature-known Middle Pleistocene specimen reads thousands of years rather than its assigned 430,000 (Lindahl 1993; Allentoft et al. 2012). Across all of them the recent-origin model accounts for the data with one founding plus bottleneck event, where the standard model requires a separate auxiliary mechanism for each. We address the principal objections, that a mitochondrial most recent common ancestor need not coincide with a population's origin, and that selection, background selection, and saturation can reconcile pedigree rates to deep timescales. These results align the mitochondrial clock, and the demographic, historical, and biochemical record around it, with a recent, creation origin rather than a deep-time one.

Keywords: mitochondrial DNA, pedigree substitution rates, hypervariable regions, mitochondrial MRCA, molecular clock, recent human origins, biblical demographic model, control region, coalescence, Neanderthal; Denisovan

1. Introduction: pedigree rates versus phylogenetic rates

In 1962 Zuckerkandl and Pauling proposed the molecular clock, the idea that mutations accumulate at a roughly constant rate and so can be used to date branching events. Applied to humans, the clock has almost always been calibrated phylogenetically: an external event, most often the postulated 6 to 7 million year human and chimpanzee divergence drawn from the fossil record, is used to fix a node, and the substitution rate is solved for from that assumed age. As Kimura (1983) framed the neutral expectation, the rate at which substitutions accumulate between species should equal the neutral mutation rate, so phylogenetic and pedigree methods should in principle return the same number. They do not, and the gap is large and persistent.

We distinguish two quantities that are often conflated. A mutation rate describes how often new variants arise per genome per generation. A substitution rate describes the rate at which heritable sequence states replace ancestral states along a lineage. Some pedigree studies instead report detectable intergenerational changes in heteroplasmy state. Because these endpoints are not identical, we identify the endpoint used by each study and treat the pooled 1/19,947 estimate as a composite pedigree-event rate. At strictly neutral sites the two converge, because although most new variants are lost to drift, the fixation probability of each new variant exactly offsets the number arising. This identity is what makes a neutral region usable as a clock, and it is central to the analysis below. As Biblical creationists we hold that humanity and the other created kinds originated on the order of 6,000 to 10,000 years ago, with a global Flood bottleneck between roughly 4,500 and 5,500 years ago in the Masoretic and Septuagint chronologies. That model predicts a rapid, near-neutral substitution signal that should place the human mitochondrial MRCA within this window. The purpose of this paper is to not only test that prediction directly against the published pedigree literature, but also against a phylogenetically calibrated rate whose calibration is the very point in dispute. But to also test the rate of fixation using the biblical timeframe and scenario including two coalescence calculations. We acknowledge at the outset that mtDNA coalescence does not by itself date the origin of humanity; we take up that objection in Section 8.

2. The clocks inside the mtDNA

The 16,569 bp human mitochondrial genome divides into a large coding region of roughly 15,447 bp and a non-coding control region, the D-loop, of about 1,122 bp. The coding region is under strong purifying selection, so it is not a neutral clock: selection continually removes variants and the apparent rate is depressed. The control region is far less constrained. Within it lie two hypervariable regions, HVR1 and HVR2. Because variation at these sites is effectively neutral (*Howell et al. 2007*), selection exerts little influence compared to the coding region, which is precisely why the hypervariable regions are the most appropriate internal clock for dating recent coalescence. Sequencing windows for HVR1 and HVR2 differ slightly between laboratories, which accounts for the small variation in reported region lengths across the studies cited here. For clarity, our coalescence and fixation calculations use the combined HVR1 and HVR2 regions. Sequence-window length does not directly enter the age equation once both the observed difference count and the pedigree rate have been measured for the same sequence window. If only the D-Loop rate is published we seek to find if all mutations were found in the HVR regions so we do not conflate or publish the wrong rate. We therefore match or standardize sequence coverage before applying a pedigree rate to an observed pairwise difference count.

3. What was predicted, and what pedigrees found

Using a phylogenetically calibrated rate, Howell et al. (1996) framed the evolutionary expectation as roughly one mutation every 590 generations across the span they examined, equivalent on their figures to about one substitution every 14,750 years. Direct pedigree measurement told a very different story. Howell noticed a substitution every 25 generations equivalent to 500 – 600 years. This was an order of magnitude faster than expected by evolution theory. Quoted directly from his paper we read: “A divergence rate of 2%/Myr extrapolates to a mutation frequency of $\sim 2.6 \times 10^{-5}$, **a 200-fold-higher rate.**”...“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”. (emphasis added)

Over time and across diverse people groups, pedigree depths and regions, studies kept observing rates an order of magnitude faster than evolutionary predictions, many times faster than the phylogenetic expectations. One of the authors of these studies recognized the implication immediately. Parsons et al. (1997) wrote: “Thus, our observation of the substitution rate, 2.5/site/Myr, is roughly 20-fold higher than would be predicted from phylogenetic analyses. 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. Even acknowledging that the MRCA of mtDNA may be younger than the MRCA of modern humans, 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.” (emphasis added)

They added that even granting that the mtDNA MRCA can be younger than the population MRCA, it remained **implausible**, on their assumptions about the geographic distribution of variation and human migration, to explain that distribution in only the last 6,500 years. We read this as a conflict between the observed rate and a prior commitment to a deep timescale, not as a defect in the measurement. The measured rate is what it is; the pedigree measurements themselves are direct observations. Their interpretation depends on the demographic and evolutionary assumptions applied. Many may assume these rates are not for determining history or ancestry but they are directly for that. In their own words... *"This observed rate has implications for both forensic identity testing and for the inference of human history from mtDNA sequences."* Parsons et al 1997.

4. Short-term and long-term rates, and the robustness of the pedigree signal

Critics frequently argue that short-term pedigree rates do not capture long-term rates. It is worth being precise about what the two terms mean. Short-term, pedigree, or germline rates are measured directly by counting new substitutions transmitted from parent to child in documented lineages or laboratory populations. Long-term, phylogenetic rates are not a separate empirical measurement; they are estimates produced by comparing assumed-related species across assumed divergence times calibrated to fossils. The long-term rate is thus the conclusion of the evolutionary model, not an independent check on it. Pedigree rates are observable, repeatable, and falsifiable; phylogenetic rates inherit whatever calibration is assumed. For that reason we treat the pedigree rate as the primary datum. Since our model falls within just a few hundred generations, we do not need to factor in phylogenetic long term rates whatsoever. The two are totally independent from one another and need not cross. So while we can recognize that there is a difference, in our model it makes no difference. So we are not treating a short term pedigree rate as a long term rate since there is no long term timeline in our model. All the problems that exist for the evolutionary paradigm regarding observed pedigree rates are just that, problems for evolution, and have no bearing on our model at all.

4.1 Placing HVR1-only studies on the combined HVR1 plus HVR2 basis

This paper looks at major pedigree studies over the last 3 decades. Some studies only published a HVR1 rate, others both while others the entire D-loop control region. Mumm et al. (1997) and Madrigal et al. (2012) specifically, report mutation rates for the first hypervariable region alone, whereas the pooled rate in this study is expressed on the combined HVR1 plus HVR2 window. To place any of these studies on the common basis, we need a defensible estimate of how much faster the full window accumulates substitutions than HVR1 by itself, and we take that adjustment directly from Árnadóttir et al. (2024). We regard it as the single most reliable anchor available anywhere in the pedigree literature. The reason is scale and resolution. Árnadóttir et al. is the deCODE germline mitochondrial study, and it is not one more small pedigree dataset but the largest ever assembled, resolving 8,199 mutations across 116,663 mother to child transmissions in 2,548 matrilineal sequences sequenced to a median mitochondrial depth above 3,000 times. Where each classical pedigree study rests on a handful of observed events, deCODE observes mutations in the thousands, which collapses the sampling error that makes the smaller studies individually noisy, and its whole genome sequencing detects and site resolves heteroplasmies directly rather than inferring them. Critically for the present purpose, the study reports separate, directly measured per site rates for HVR1 and HVR2 rather than a single control region average (their Table S3): 3.26×10^{-5} mutations per site per generation for HVR1 (95% CI 3.09 to 3.44×10^{-5}) and 4.07×10^{-5} for HVR2 (95% CI 3.86 to 4.28×10^{-5}). The difference between the two regions is therefore measured, not assumed: HVR2 mutates 4.07 divided by 3.26, or 1.25 times as fast per site as HVR1. The same figure is visible in the raw counts, where HVR2 carries more mutations (1,418) than HVR1 (1,353) despite being the shorter segment (299 versus 356 base pairs), and it is corroborated in direction by the other studies that measured both regions separately, including Hühne et al. (1998), Howell et al (1996, 2003), Parsons et al (1997), Cavelier et al. (2000) but only as somatic CD34+ cell data, Santos et al. (2005), Soares et al. (2009) Heyer et al (2001) Morovvati et al. (2011) a common theme across multiple independent studies spanning decades. We use only this ratio from deCODE, not its absolute rate. Each HVR1-only study continues to supply its own directly observed HVR1 rate and its own generation interval, and deCODE contributes nothing but the relative weighting between the two segments. The conversion is done in per site terms so that every step is transparent. A study's observed HVR1 rate per site, call it r_1 , equals its events divided by its transmissions divided by its HVR1 length.

We impute the HVR2 rate for that same study as $1.25 r_1$. Over the combined 610 base pair window, taken as HVR1 of 342 base pairs (positions 16024 to 16365) and HVR2 of 268 base pairs (positions 73 to 340), the expected events per transmission are then $r_1 \times 342$ plus $1.25 r_1 \times 268$, which equals $r_1 \times 677$. In other words, because HVR2's 268 sites each mutate 1.25 times as fast, they contribute the equivalent of 335 HVR1 sites, so the 610 base pair window behaves like 677 HVR1 equivalent sites. The combined basis interval is the reciprocal of that rate. This procedure replaces the cruder assumption of equal segment lengths with the measured lengths, and the result is insensitive to the one quantity that is imputed rather than observed. Propagating the deCODE 95 percent confidence interval on the ratio, approximately 1.12 to 1.39, through the effective length gives a range of 642 to 715 HVR1 equivalent sites around the central value of 677, a spread of roughly plus or minus five percent on any converted interval. Published estimates of the HVR2 to HVR1 ratio vary in magnitude, and occasionally in direction, across datasets, which is precisely why we anchor on the deCODE value for its precision and report a sensitivity range rather than treating any single smaller study as superior or definitive. The combined basis rates for the two HVR1-only studies are therefore governed by their own observed HVR1 events and by a ratio measured across more than a hundred thousand transmissions, and they do not depend on any assumption we cannot support with data.

The use of an independently estimated relative regional rate to place an incompletely assayed sequence window on a common analytical basis is neither unprecedented nor methodologically ad hoc. Indeed, many subsequent mutation rate studies have employed more explicit relative-rate adjustments. Soares et al. (2009) empirically derived a coding-to-control-region mutation ratio of 1.57; Cox et al. (2012) imported that value as “a *mutation rate scalar of 1.57 coding region mutations to every control region mutation*,” Hallast et al. (2016) scaled a whole-mtDNA rate to the coding region using the same ratio, and Batini et al. (2011) expressly adapted an external mitochondrial calibration “to the sequence range considered” (coding-region) in their analysis. Other pedigree rate comparisons have likewise converted heterogeneous published estimates to common sequence-length, generation time, or per-site units (Santos et al., 2005; Henn et al., 2009; Connell et al., 2022). The present procedure is more conservative than importing an external absolute molecular clock: Mumm et al. and Madrigal et al. continue to supply their own observed HVR1 mutations, transmissions, and generation intervals, while Árnadóttir et al. (2024) contributes only the dimensionless HVR2-to-HVR1 weighting factor. No additional HVR2 mutations are represented as having been observed in the original pedigrees. The resulting values are explicitly identified as standardized, extrapolated HVR1-plus-HVR2 estimates and are accompanied by a sensitivity analysis. This is therefore a transparent partition-rate conversion grounded in the largest directly measured human mitochondrial pedigree dataset, not the manufacture of an unobserved mutation rate.

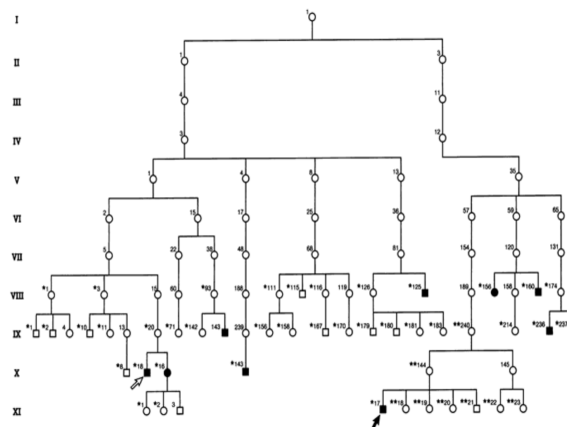
Defense Science Board (1995)

The 1995 Defense Science Board report on DNA identification shows us that AFDIL obtained a substitution rate we will investigate. The report first states the conventional expectation, an evolutionary fixed rate of roughly one control-region difference per 300 generations, about one difference every 6,000 years, then records that AFDIL had already observed at least two examples of fixed sequence differences between a combined 46 explicitly mother–child CEPH comparisons with 30 routine casework comparisons. (*Defense Science Board, 1995, p. 18*). In the best-documented family the daughter differed from her mother at two nucleotide positions while the mother's two sons did not differ, and the maternal relationship was independently confirmed with 12 DNA markers, including 8 RFLP loci (*Defense Science Board, 1995, p. 19*). The report is explicit that these are fixed sequence differences and not low-level heteroplasmy, which makes the verified two-position family a confirmed instance of homoplasmic control-region change across a single maternal transmission. Read conservatively as single-generation events, a minimum of three fixed substitutions across those comparisons implies on the order of one control-region substitution per 10 transmissions. We take three as the working minimum, rather than two, because the report speaks of at least two examples of mother-child discordance and then describes only one of them, the family carrying two differences, so the second discordant family must contribute at least one further substitution, giving two plus one. The report then supplies a second, independent way to bound the rate. AFDIL notes that it also examined 46 controlled CEPH mother-child comparisons and found no differences at all, and it

gives its own preliminary figure, drawn from a larger study spanning roughly 300 generations, as closer to one difference per 50 generations (*Defense Science Board, 1995, p. 19*). Because the CEPH series is a distinct body of data from the casework, and neither is the material Parsons et al. (1997) would later analyze, the two can be pooled as one AFDIL dataset. Pooling the 46 CEPH comparisons with the roughly 30 casework comparisons gives about 76 maternal comparisons carrying a minimum of three fixed substitutions, or at least **one substitution per 25.3 comparisons**. We adopt that combined figure, approximately one per 25, as the rate this source contributes to the analysis, with the casework-only value of about one per 10 as the fast rate bound and AFDIL's own preliminary one per 50 as the slow rate bound. All three sit far above the phylogenetic estimate of one per 300 that the same report cites. Derivation: 46 CEPH comparisons plus approximately 30 casework comparisons give about 76 maternal comparisons; with a minimum of 3 fixed substitutions, 76 divided by 3 equals 25.3, hence at least one substitution per 25.3 comparisons.

Howell, Kubacka, and Mackey (1996)

They add together 45 members of the TAS2 family whose complete D-loop was sequenced and 4 additional individuals from English families who carry the TAS2 D-loop haplotype, for a total of 49 transmission events over 11 generations, with exactly 2 new germ-line D-loop substitutions detected, both were located in hypervariable region II (*nucleotide positions 152 and 195*), and none in hypervariable region I. Two mutations divided by 49 transmissions gives the observed rate of **one substitution every 24.5 transmissions**, which the authors round to roughly 1 per 25 generations.



Mumm et al. (1997)

Mumm and colleagues amplified a 443 bp fragment of the mitochondrial D-loop by nested PCR and compared sequences across two related eastern Missouri kindreds with X-linked recessive hypoparathyroidism, over a 367 bp analyzed region of HVR1. Although the study was designed to establish common ancestry rather than to measure a mutation rate, it captured one clear de novo maternal-line event, the A to G transition at position 295 that arose in a branch of the Whyte kindred and segregated through subsequent generations. Counting that single event across the maternal transmissions observed in the pedigree gives **one substitution every 59 generations in HVR1**, or 4.62×10^{-5} substitutions per site per generation across the 367 bp region. Applying the deCODE conversion set out in Section 4.1, the imputed HVR2 rate is 1.25 times this HVR1 rate, and the combined 610 base pair window carries the equivalent of 677 HVR1 sites, so the combined rate is $4.62 \times 10^{-5} \times 677$, or 0.0313 events per transmission. This equates to **one substitution every 32.0 generations** on the combined basis, a very common theme throughout the pedigree literature. This is the first study where we had to implement an HVR2 rate since only an HVR1 rate was published, the HVR2 calculation as stated earlier is imputed from the deCODE ratio rather than measured by Mumm. This is a conservative approach as always and will favor the slow end through implementation.

Soodyall et al. (1997)

Soodyall and colleagues observed **zero new fixed substitutions across 108 maternal transmission events** in their Tristan da Cunha pedigrees. Because no substitution was observed, the dataset does not provide a finite positive-event estimate in generations per substitution. The authors nevertheless expressed the result as an upper-bound constraint, reporting that the mutation rate was no greater than approximately **one new mutation every 36 transmissions**. This value corresponds closely to the conventional rule-of-three approximation for a zero-event dataset, $3/108$, and should therefore be interpreted as an approximate upper confidence bound rather than as an observed rate.

Parsons et al. (1997)

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. Scanning both hypervariable regions (HVR1 & HVR2) 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)."

Pedigree transmission depth is not directly mentioned for all pedigrees but the theme quoted is "predominantly mother:child, grandmother:grandchild, or sibling pairs". This is referring to only dyad and triad depth, so mostly just two and three generations deep pedigrees were cataloged, especially in the AFDIL samples. This lack of depth is made up for in sheer number of test subjects. 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). Parsons 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 within 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 published as 10 fixed substitution differences after 327 generational links or **one substitution every 33 transmissions**.

Table 1 • Summary of sequence comparisons and observed mutations					
Sample source	mtDNA lineages	Generations	Mutations	Rate per generation	Rate pooled by type
AFDIL family references	73	121	7	0.0578 1/17	blood samples 0.052 1/19
'Oxford' families	5	32	1	0.029 1/32	
Amish families	40	80	1	0.013 1/80	cell lines 0.011 1/87
CEPH families	16	94	1	0.011 1/94	

Figure 1. Taken from Parsons et al 1997 study showing results from all samples. Samples resulted in a variation of ranges from extremely fast 1/17 – to extremely slow 1/94. You can observe in the Table 1 chart in the Parsons paper on page 2, under section; Observed substitutions. Note that the single generation AFDIL family references were single generation and obtained the fastest rates, the triad studies obtained slower rates. The Oxford families resulted in 1/32 while the Amish families mutated slowly at just 1/80.

To illustrate the implications of this faster rate, Parsons et al. compared it with a previously published evolutionary calibration of 0.118 ± 0.031 substitutions per site per million years (Stoneking et al. 1992), which had produced a point estimate of approximately 133,000 years for the global human mtDNA MRCA. Based on the rounded statement that their rate was "roughly 20-fold" faster: 133,000 ÷ 20 = 6,650 years. Parsons et al.'s published value of approximately 6,500 years reflects their rounded characterization of all pedigree rates as roughly twentyfold faster. Parsons did not independently produce the 133,000-year date from his pedigree data but rather as stated in his paper, used an evolutionary date from an earlier paper by Mark Stoneking, Stephen Sherry, Alan Redd, and Linda Vigilant (1992) titled *New Approaches to Dating Suggest a Recent Age for the Human mtDNA Ancestor*. Parsons' listed this study as reference 6.

To obtain an exact MRCA date using the published substitution rate directly gives $133,000 \times (0.118/2.5) = 6,277.6$ years, or approximately 6,278 years. His published rate of approximately 1/33 generations was not independently derived from a single pedigree; it was obtained by pooling the 10 observed substitutions across all the 327 generational links from all four sample sources. *Parsons'* was explicit about this. He stated that *"lacking a resolution to the apparent different rates observed for blood versus cell line samples, we will adopt the pooled value, 0.03 per generation, for the purposes of consideration and comparison."* He discusses both rates directly, states that the discrepancy between them is statistically significant, and then chooses the conservative combined slower figure specifically. We point to this study to show you directly how different pedigrees can result in different rates. This is extremely important to recognize and will continue with this theme throughout the paper.

Macaulay et al. 1997

An attack of Parsons and Howell's work by Macaulay et al. quickly followed. He specifically targeted four things. Hot spots, variable sites, fixation and that the rate fails archaeology based on radiometric dating. All arguments were defended soon after by Howell and Mackey in Genet. 61:986-990, 1997, Titled: Reply to Macaulay et al.

The four attacks and how Howell answers them

1. **"It's just HVR2 hot spots."** Macaulay says the fast rate is inflated because the two new mutations landed in HVR2, a hotter region. Howell answers that new mutations are actually spread across both HVR1 and HVR2 (Parsons' 10 substitutions, 3 in HVR1; Torroni; Kocher and Wilson), and even the HVR1-only comparison is still tenfold faster. So hot spots cannot *"swamp out"* the signal. A vivid phrase he uses to dismiss the idea that HVR2 hot spots inflate the rate (Image 2, right column).
2. **"They're all higher-than-average sites."** Macaulay leans on twin data (Bendall 1996) to say the fast mutations sit at unusually variable sites. Howell calls the phrase misleading: *"higher than average"* is measured against all sites, most of which never mutate, so almost any real mutation clears that bar. He adds that Bendall's spectrum was unremarkable and that they omitted twin pair 1, whose inclusion would have made the rate faster still. Howell and Mackey rejected the notion that a handful of rapidly mutating HVR2 sites could *"swamp out"* the signal, concluding instead that *"there is a real discrepancy"* between the two rates
3. **The fixation argument.** Howell concedes the pedigree rate legitimately exceeds the phylogenetic rate because many new mutations never reach fixation in the whole population. He walks through mtDNA segregation (the triplasmic female IX-240, figure 1) to show heteroplasmy behaves unpredictably, so a substantial fraction of new variants are lost before they ever fix. This by no means is damaging to the substitution rate, since the region is effectively neutral.
4. **"It fails the archaeology."** Macaulay uses Polynesian and Cook Islands sequences to argue the fast rate gives coalescent dates that fit the archaeological record worse. Howell says there is too little methodological detail to even assess that claim, and that it is premature to expect a clean monotonic decline of rate with time depth. He states: *"the lack of experimental and analytical details"* a jab at the rigor of Macaulay Cook Islands analysis (Image 4, right column). Macaulay never made a follow up paper or attempted rebuttal.

Howell concludes, *"there is a real discrepancy"* between pedigree and phylogenetic rates. This is found in the closing thesis (Image 4, right column). A discrepancy that even to this day almost 30 years later has never closed. Howell and Mackey are a good confirmation that the Parsons rate is both rapid and real and was defended in AJHG, without rebuttal. The fixation argument actually strengthens our case, since a fast substitution rate plus incomplete fixation is exactly what a short, recently bottlenecked history predicts. So few fixed differences with such a fast rate and not a lot of time is exactly what the genetic evidence shows. The following year Parsons decided to do a re-test because of the controversy.

Sigurðardóttir et al. (2000) *The Anomaly*

Sigurðardóttir et al. (2000) is overshadowed as an accurate study and mutation rate because the estimate is too imprecise to carry weight as stated by the authors themselves and has since been superseded by a much larger pedigree study of the same population in 2024 by Árnadóttir, E. R., & Helgason. Sigurðardóttir's estimate could not therefore be weighed the same since the same people group tested later resulted in totally different outcomes, and it does not carry the same precision of the larger pedigree that came after it by the same team who did this smaller pedigree 24 years earlier with Sigurðardóttir. Our reservations are methodological, not biased and confirmed by the later Árnadóttir et al., 2024 validating that the earlier Sigurðardóttir study was incorrect and an outlier. Again, the same research group, working in the same Icelandic population and including the same senior author (Agnar Helgason), published a pedigree study based on 116,663 mother to child transmissions across 2,548 matrilineal lines and found thousands of mutations. That is on the order of 165 times the transmission count and more than 2,700 times the number of observed mutations, and it documents extensive hypermutability concentrated in the D-loop, including taking into account parallel mutations, gender, heteroplasmy, disease, contamination, selection and more. The technology 24 years later has greatly improved and we choose to rely on the modern day large-pedigree estimates of the same people and treat the older Sigurðardóttir et al. (2000) as a historically important but low-precision precursor, now subsumed by the deCODE pedigree data of 2024 by Árnadóttir, E. R. et al. Who took Sigurðardóttir's observed published rate of only one substitution every 235 generations all the way down to **one substitution every 38 generations** in the same group tested. This goes to show you how far off smaller studies can be from true. This is why we refer to larger pedigrees whenever we can and especially more diverse people groups and populations to get us a broader picture such as Parsons.

Santos et al. (2005)

Azorean families produced 321 transmissions, across 48 lineages covering the full D-loop region which includes both HVR1 + HVR2 and detected 13 new mutations (*11 substitutions + 1 possible deletion + 1 deletion*), affecting 12 distinct positions in 321 transmissions. That gives a raw familial rate around 1 mutation every 24.7 generations. The slower figure the authors also reported came only from filtering: they reclassified almost half of the observed variants as somatic, and excluded male-line transmissions. They were candid that the line between germline and early-somatic events cannot be drawn with certainty and that their corrected rate is therefore a lower bound, stating; *"...in these families the reported heteroplasmy may have resulted from somatic mutations that occurred in the individuals themselves, or alternatively, it may have resulted from germ line mutations that occurred in the oocyte from which the heteroplasmic individuals developed."* This is important because their calculation counts 11 substitutions in 321 transmissions, approximately **one substitution per 29 transmissions**, after filtering out two. All 11 positions were inside the HVRs: 16086, 16150, 16189 (twice, two separate families), 16264, 16309, 64, 150, 152, 215, 238. Resulting in a ~2.0/site/Myr mutation rate, or a ~4.1/site/Myr divergence using their 29.5 generation time or at a 20 year generation time, and like Parsons et al, it becomes a rate of ~2.81/site/Myr. Their filtered estimate of 0.2415 mutations/site/Myr is therefore not a directly measured rate. It is a model-dependent filtered rate based on assumptions and removal of males (that filtered version gives 1/53.5, still a very fast rate. Although Santos et al. favored a slower fixation-corrected rate, they acknowledged that several mutations classified as somatic could instead have originated in the germline, that the actual probability of heteroplasmic fixation could not be truly be estimated from their data, and that their neutral fixation model failed to explain at least one observed family. Consequently, the heavily filtered rate was a model dependent projection rather than a directly measured rate, and the data do not exclude a rate substantially closer to the raw pedigree estimate, which they acknowledge and is why they kept the raw rate as their upper bound published rate. We agree since it aligns with decades of independent other studies, but not their filtered rate and we regard their raw rate as the appropriate estimate, while noting that the classification of some heteroplasms are assumed to be somatic and the removal of males from the results is a genuine methodological judgment, one we cover in section 6. The fact is after removing one deletion and another possible deletion left Santos with 11 substitutions across 321 transmissions over 973 bp of the D-loop, a rate of **one substitution per 29 generations**, although because of uncertainty about removing true mutations including other variables the upper confidence-limit rate of 0.054 mutations per generation would equal: $1 \div 0.054 = 18.52$ or one substitution every 19 generations.

Moreau et al. (2009, 2011)

Several features make this pedigree-derived estimate unusually transparent. Moreau et al. reconstructed the maternal genealogies using the BALSAC population database, the Early Quebec Population Register, marriage repositories, and family dictionaries. Their sequencing covered HVS-I positions 16069–16383 and HVS-II positions 58–370. Sequence calls were verified from both strands by two independent analysts and were additionally screened for phantom mutations and reticulations in the haplotype network. Supplementary Table S10 identifies JCL377 as the only haplogroup-A sample carrying np 16343, whereas DS187, GEL407, GEL411, and GEL412 share the otherwise identical haplotype without that position. Because np 16343 lies within the sequenced and comparatively analyzed HVS-I interval and is recorded as a nucleotide difference rather than a length variant, it cannot be attributed to instability in an HVS-II poly-C tract. This is a real, consensus-level substitution captured within a deep documented pedigree. Counting every mother-to-daughter link from their most recent common maternal ancestor down to each sampled participant, and tracing each terminal branch all the way to the living carrier rather than to any dated marriage, takes us back to the year 1740. The result is an empirically reconstructed control-region HVR rate of **one substitution per 13 generations**. The unique 16343-bearing terminal lies at the end of a six-transmission branch, localizing the net sequence difference to that branch under the most parsimonious reconstruction. Moreau et al. did not compute this figure, yet they explicitly read these singleton differences as mutations arising within their own pedigrees and attributed them to the high mutation rate of the mitochondrial D-loop. Their data do not merely permit a fast maternal clock. Their data do not merely permit a rapid maternal clock. They document a consensus-level sequence difference across a multigenerational pedigree whose maternal-transmission exposure can be reconstructed directly from the published genealogy.

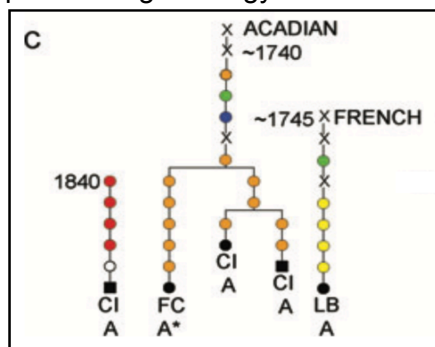


Figure 2. Haplogroup-A maternal genealogies reproduced from Moreau et al. (2011, their Figure 2C Left side showing the exact pedigree). Five sequenced haplogroup-A carriers are shown, three of whom, including the uniquely altered A* carrier, are connected within the central maternal pedigree. Black symbols represent sampled participants; circles and squares denote females and males, respectively; FC, CI, and LB denote French-Canadian, Channel Islander, and Loyalist identification; and the lowest letter identifies mitochondrial haplogroup A. The asterisk marks the singleton haplotype containing np 16343. Colors identify recorded places of marriage, while an “x” indicates an unknown location. For the present analysis, only the three connected carriers are used.

Madrigal et al. (2012)

Madrigal and colleagues reconstructed thirteen deep-rooting pedigrees from Atenas, Costa Rica, using Church Chancery and National Document Register records, with most recent common ancestor birth years spanning 1600 to 1876 and several lines tracing to colonial founders in the 1600s. After excluding two pedigrees flagged for possible adoption, they counted seven mutations across 273 maternal transmissions in HVR1. They report two HVR1 estimates, one counting all apparent mutations and a more conservative filtered figure of 25.3×10^{-6} substitutions per site per generation, with the all substitutions rate sitting roughly 2.8 times above their conservative one. Madrigal et al. (2012) explicitly noted that their maximum pedigree estimate was “**very close to the highest value ever reported (Parsons et al., 1997)**,” with rates of 2.51 and 2.50 substitutions/site/Myr, respectively. They specifically named Parsons which their rate matched even though their pedigrees were multi-generational. **Madrigal et al: 2.51 substitutions/site/Myr & Parsons et al. 1997: 2.50 substitutions/site/Myr.** The HVR1 only rate is therefore 7 over 273, or 0.02564 substitutions per transmission, equivalent to **one substitution every 39 generations**. You may be wondering how a rate faster than Parsons who obtained 1/33 is published at 1/39. This comes down to base pair size and window. Parsons tested both HVR1 and HVR2, Madrigal only tested HVR1. When windows are matched, the rates equal out to being almost the same with Madrigal still being faster than Parsons in the end. Applying the deCODE conversion set out in Section 4.1, the imputed HVR2 rate is 1.25 times this HVR1 rate, and the combined 610 base pair window carries the equivalent of 677 HVR1 sites, so the combined rate is $7.12 \times 10^{-5} \times 677$, or 0.0482 events per transmission. This is **one substitution every 20.7 generations on a combined HVR1 and HVR2 basis**. As with Mumm et al, the HVR2 term is imputed from the deCODE ratio rather than measured by Madrigal since this study requires both.

In sequenced transmission terms, the Costa Rican pedigree had many deep lineages. The deepest observed pedigree in the paper was **pedigree 13**, where 2 sequenced individuals are connected through 15 meioses. Because only 2 subjects were typed, all 15 of those mother-to-child links fall on the single path between them, so this is the deepest genuinely sequence-anchored span in the study: one uninterrupted 15-transmission chain bracketed by sequenced tips at both ends. This is a single chain of 7 – 8 generations back.

Pedigree 8 is right behind it (2 typed, 14 meioses, MRCA 1750) both pedigrees are visible on the right. The paper's own raw table holds an even faster figure; a durable result sits inside the retained pedigrees themselves. Pedigree 5 recorded one mutation across eighteen meioses, a rate of 1/18, closely matching the AFDIL blood-sample estimate of approximately 1/17 in Parsons et al 1997.

Madrigal's **pedigree 5** results here are not a marginal case flagged for adoption; it survived every filtering step the authors applied and remains part of their own maximum estimate. What makes it notable is depth. Table 1 in their paper gives this pedigree's most recent common ancestor a birth year of 1834, and unlike the shallow single generation pedigrees like most of Parsons, this one is directly tracked over eight generations, and this pedigree was confirmed from multi generation Church Chancery genealogical records rather than a single mother-child comparison, so the 1/18 figure reflects mutations accumulating and being directly observed across a genuine multi-generation lineage rather than a dyad or triad study which are claimed to have been inflated by a shallow pedigree depth. Two additional pedigrees reported in the raw table produced faster pedigree-specific estimates: pedigree 10 at approximately 1/9 and pedigree 11 at 1/10. Together, these fast lineages illustrate the same pattern Heyer's fast-site model documents at the population level, while pooling many pedigree studies into a single estimate lowers the reported rate when other included studies observed few or no events. The critics of pedigree studies who have said that deep rooted pedigrees slow the rate down now have multiple pedigrees in different studies turning in rates as fast as anything *Parsons*, regardless of his pedigree depth.

The study pedigree transmission depth overall: across all 19 pedigrees, MRCA birth years in Table 1 of their paper range from 1600 (pedigree 3) to 1876 (pedigree 19). That is a depth range from roughly 14 generations (pedigree 3) down to about 4.6 generations (pedigree 19) using the study's 28.3 year generation time interval.

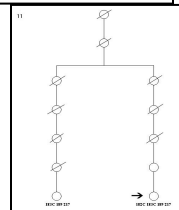
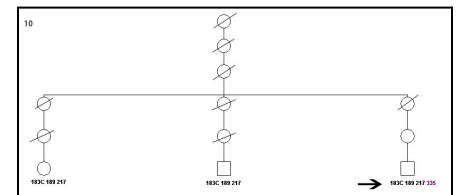
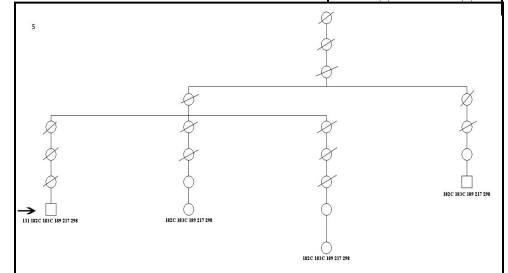
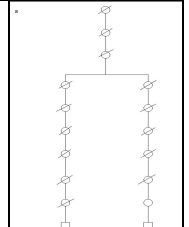
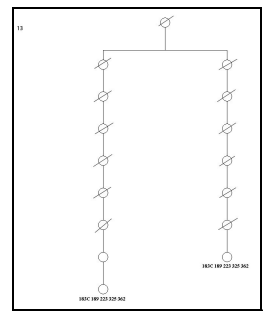
Pedigree 5 (the 1/18 result): MRCA born 1834, 18 meioses across 4 typed subjects, 1 mutation at site 16,131. Pedigree meiosis depth from 1834 and encompasses a total of **8 generations**.

Pedigree 10 (the 1/9 result): MRCA born 1846, 9 meioses across 3 typed subjects, 1 mutation at site 16,335. Depth from 1846 to 2005 is about 159 years, **6 generations**. (Top image on the right)

Pedigree 11 (the 1/10 result): MRCA born 1802, 10 meioses across 2 typed subjects, 1 mutation at site 16,182. Depth from 1802 to 2005 is about 203 years, **7 generations**. (Bottom right).

Wei et al. (2019)

Wei et al is a Chinnery lab's study built on the 100,000 Genomes Project and the NIHR BioResource, analyzing 12,975 whole mitochondrial genomes, with a population-level validation set of 40,325 individuals. The transmission analysis consisted of 1,526 mother-offspring pairs. Deep whole-genome coverage was performed, the control-region rate implied by their counts is 7.64×10^{-5} per site per generation (95 percent CI 6.39 to 9.06×10^{-5}), which over the entire 676 bp HVR1 and HVR2 window is approximately **one substitution every 19 generations**, comparable to what was observed and documented in pedigrees by Parsons, Brandstätter, some Madrigal pedigrees. Since all mutations were observed in the typical 610 bp locations, the rate stays the same. Wei's rate is therefore another, powerful confirmation that observed rates vastly exceed phylogenetic ones.



Connell et al. (2022)

The researchers sequenced the complete mitochondrial genomes of 225 individuals drawn from 45 maternal lineages in the Norfolk Island population, representing 345 maternal transmission events. The sampled individuals were drawn from the most recent four generations of a much larger documented Norfolk Island pedigree. Complete mitochondrial genomes were sequenced using next-generation sequencing, with candidate mutations subsequently confirmed by Sanger sequencing. Across the complete mitochondrial genome, Connell et al. identified nine mutation events, consisting of two homoplasmic transitions and seven cases of point heteroplasmy. For regional only analysis, the authors defined HVI as positions 16024–16383 and HVII as positions 57–371. Their published pedigree estimates were 1.201 mutations/site/Myr for HVI, 0.343 mutations/site/Myr for HVII, and 0.801 mutations/site/Myr for the combined HVI/HVII regions, using a generation interval of 26.9 years. All detected mutations, including heteroplasmic variants meeting the authors' inclusion threshold, were incorporated into these estimates. Inspection of the underlying pedigree mutation table identifies five HVI/HVII point-mutation events among the 345 maternal transmissions: A16280R, T146Y, A16247G, C16344Y, and C16320Y. These positions also fall within the narrower combined HVR1 and HVR2 sequence interval employed in the present analysis. Consequently, the observed event frequency can be calculated directly from the raw pedigree data without extrapolating the published per-site rate: 5 observed HVR point-mutation events ÷ 345 maternal transmissions = 0.01449 events per transmission or, reciprocally, 345 ÷ 5 = 69 maternal transmissions per observed HVR point-mutation event. Connell et al. therefore provide an observed combined HVR1/HVR2 pedigree event rate of approximately one point-mutation event every 69 maternal transmissions (1/69). Connell et al.'s comparatively slow HVR event rate must also be interpreted in light of their deliberately conservative analytical criteria. The analysis excluded multiple high mutation rate mitochondrial length-variation sites. Many known hotspots were completely disregarded. There is a reason they are an extreme outlier within HVR rate studies. All in all Connell et al dropped the rCRS indel hotspots (309, 455, 463, 573, 960, 5899, 8276, 8285, 16193), plus 16180 to 16183, and the HVII tracts (302 to 316). Connell threw all of it out rather than try and resolve it. The comparatively slow value of approximately 1/69 became his filtered published rate.

Árnadóttir, Helgason, et al. (2024)

Based on 116,663 mother-child transmissions, 8,199 mutations were detected. The largest matriline contained 2,056 transmissions, linking 1,051 sequenced individuals traced back to an ancestor born around 1520. That is the largest matriline by transmission count, not necessarily the deepest one by generations. The actual depth range across the whole study is wider: the birth year of the matriline most recent common ancestor ranges from 1410 to 1995, with a mean of 1834.6 and a median of 1840; generation length ranges from 10 to 50 years across matriline, with a mean of 29.3 and a median of 30. So the true deepest-by-date matriline traces back to 1410 for the MRCA. Regarding pedigree transmission depth, this is harder to pin down, individually-documented, generation counted chains of transmission events is given in their study Table 1: Transmissions per matriline (*tree size, all 2,548 matriline, Table 1*): mean 45.8, median 22, range 1 to 2,059, IQR 5 to 53. It is a size measure, not a depth measure. Branch depth in generations (Table S10, the deep-pedigree subset): across 53,041 informative relative-pairs, the deeper individual in a pair sits a mean of 4.7 generations (median 5) back from the shared ancestor, with pairs distributed all the way from 1 to 7 generations deep. About 57% of pairs reach 5+ generations deep, 36% reach 6+, and 15% reach the full 7 generations deep. So the average runs 5 generations and max 7.

We do not have a true raw rate count, everything that is published is after filtering, they note that 1,667 mutations from 1,324 individuals were removed based on the perception that may have all had hematopoiesis (*the process by which the body produces new blood cells*), and a further filtered 143 mutations were associated with contaminated samples and also removed. The CH filter probably removed genuine mutations, but the number cannot be determined from the published data. It was deliberately set to be a strong filter designed to sacrifice some assumed genuine mutations in exchange for reducing blood-derived somatic false positives. This means that in actuality, over 10,000 true mutational events occurred and were observed before these exclusions; the number of genuine germline mutations among the excluded calls cannot be determined so their count is

conservative. That is not all, *Árnadóttir et al.* also excluded known hotspots. They excluded 30 HVR bases altogether: The 13 bases from HVR-III poly-C tract, plus 15 bases from HVR-II and 2 bases from HVR-I. These regions contain known hot spots but were set aside because of mapping problems. Just note that even after all this filtering and accounting for the three unit germline bottleneck effect, they still published a D-loop rate of **one substitution every 38 generations**, and for HVR1 & HVR2 combined rate of **one substitution every 42 generations** (1/42). Recall that this is the same Icelandic people group tested in 2000 by *Sigurðardóttir et al* which then only produced a rate of three substitution after 705 transmissions or one substitution every 235 generations. Meaning, this study is a replacement of that older *Sigurðardóttir et al.* (2000) limited study.

5. Estimating a high end pooled rate in the mtDNA hypervariable regions of the D-Loop

Several studies fall into the category of directly measured pedigree rates, while a smaller set rely on fixed, consensus-level substitution counts alone. These fixed-difference studies are chosen because their inclusion rule matches the diversity data they are tested against: Budowle and Krings report fixed, consensus-level pairwise differences, so the rate must be built from studies that counted exactly that. The goal of this is to find the high mean among pedigree studies to test against the most diverse people on earth. We will later look at the global average but this test is required. We pooled together 4 of the higher end results and ignored all dyad and triad studies. Meaning, all shallow depth studies like *Parsons*, *Ding* and the *Defense Science Board/AFDIL* are out of the pool. The set we pool for the primary high rate average is: one substitution per 13 (Moreau et al. 2009), per 20.7 (Madrigal et al. 2012), per 24.5 (Howell et al. 1996), and per 29 (Santos et al. 2005), each is anchored to consensus-level or fixed sequence differences and full deep genealogical pedigrees.

The unweighted harmonic mean of these four fixed-difference datasets is 4 divided by the sum of their reciprocals, $4 \div (1/13 + 1/20.7 + 1/24.5 + 1/29)$, which equals approximately **one substitution every 19.947 generations or 1/20**. We adopt this as the pooled test rate throughout the coalescence calculations that follow. It is important to be exact about what this number is and is not. It is a pooled high-end mean of various pedigree datasets: a high, upper-portion average of the observed pedigree rate distribution in all cases but one (Madrigal) where we had to reconstruct a hypothetical HVR2 rate using *Arnadottir's* 2024 data since no HVR2 rate was tested or published. It is not the fastest observed rate, and it is not a global population mean. Specific individual pedigrees in the literature run considerably faster (Puerto Rican lineage in Madrigal et al. 2012, pedigree 10, at 1/9 and Pedigree 11, at 1/10) and considerably slower (the Amish pedigrees in Parsons et al. 1997, at 1/80 generations), and the population-level central rate is more in the middle of this range, as developed in Section 11. The pooled high-end mean is the value that lets us ask a specific, falsifiable question: is the upper portion of the observed pedigree distribution fast enough to generate the deep end of observed human diversity within the biblical windows? That is the test the rest of this section runs.

5.1 The neutral fixation clock and the adopted rate

Under Kimura's neutral theory the long-term substitution rate along a lineage equals the underlying neutral mutation rate, because neutral variants fix at the rate at which they arise (Kimura 1968, 1983; Zuckerkandl and Pauling 1962). The mitochondrial control region is not only neutral, its hypervariable segments carry sites that mutate well above the genomic background (Howell et al. 2007), yet across a few hundred generations those sites remain far below saturation, which is what licenses the Poisson treatment used throughout this section.

We adopt as our pooled test rate the unweighted harmonic mean of the four HVR-compatible pedigree estimates that rest on fixed, consensus-level differences: Moreau et al. (2009, 2011), Madrigal et al. (2012) at 1/20.7, Howell et al. (1996) at 1/24.5, Santos et al. (2005) The unweighted harmonic mean is four divided by the sum of the four reciprocals, $4 \div (1/13 + 1/20.7 + 1/24.5 + 1/29)$, which equals 19.947.

Even though we will be precise throughout the study, the pooled rate is approximately one substitution per 20 generations, or 0.0501 substitutions per generation across the combined HVR1 and HVR2 window; distributed over 610 base pairs this is approximately 8.22×10^{-5} substitutions per site per generation. The generation interval throughout the principal modern-human calculations is 20 years.

We describe this pooled value as a high-end mean of the pedigree datasets rather than as a single universal rate or or simple average, and we do not attach a plus or minus one generation sensitivity band to it, because the meaningful uncertainty here is the real dispersion of the underlying studies, not a symmetric interval around the pooled value. That dispersion is wide: the four fixed-difference estimates alone range from 1/13 to 1/29. This is deliberate. The pooled high-end mean is a high, upper-portion average of the observed pedigree distribution, chosen so that the numerator of the clock, the substitutions counted per generation, is kept on the same footing as its denominator, the fixed consensus-level differences counted in the diversity databases of Section 5.6. It is not the fastest observed rate, and Sections 5.4 to 5.10 test what the upper portion of the observed pedigree distribution predicts for the deep end of human diversity, not what a single tuned value can be made to yield.

5.2 The demographic scenario and chronological windows

Creation is placed at roughly 6,000 years before present on the Masoretic chronology and 7,600 on the Septuagint. A global Flood bottleneck is placed between roughly 4,500 and 5,500 years before present. At 20 years per generation these dates give the windows in Table 1. The post-Flood world descends, mitochondrially, from three founding lineages carried by the wives aboard the Ark, whose divergence from one another traces back through the pre-Flood generations to the first woman; the bottleneck reset the number of surviving lineages to three but did not reset the clock.

Event and chronology	Years BP	Generations
Flood, Masoretic	4,500	225
Flood, Septuagint	5,500	275
Creation, Masoretic	6,000	300
Creation, Septuagint	7,600	380

Table 1. Chronological windows at 20 years per generation.

According to the scriptural account, Adam and Eve were the first created humans, living in the very recent past relative to evolutionary timescales. They were commanded to be fruitful, multiply, and fill the earth. The pre-Flood population is not recorded, but it grew until it was judged and reduced, in a single generation, to the eight people aboard the Ark. Of those eight the mitochondrial record is carried by the women alone, because mtDNA is transmitted only through the maternal line. Noah's three sons carry their mother's mtDNA but do not pass it on; their wives, three women who married into the family from the wider pre-Flood population, carry three mitochondrial lineages of their own. The post-Flood world therefore descends, mitochondrially, from three founding lineages, not from a single Ark-borne haplotype. This detail is decisive for what follows. Because the three women were not sisters but unrelated wives, their mitochondrial lineages had already diverged from one another before the Flood, and that divergence traces back through the pre-Flood generations to the first woman, the mitochondrial ancestor of all humanity. The Flood bottleneck reset the number of surviving lineages to three; it did not reset the clock, and it did not erase the differences the three women already carried. We can therefore read past the three founders to the deepest human split. After the Flood the population grew again, and within roughly seven to eight generations the account places the dispersal from Babel, when a united mankind was divided and migration spread across the earth. This is the demographic scenario against which we test the fixation record: a recent origin, a single-generation bottleneck to three mitochondrial lineages, and a rapid expansion beginning shortly afterward.

5.3 Expected accumulation under lineage substitution accumulation

The word fixation must be used with care. In its strict population sense a variant is fixed when it reaches one hundred percent frequency, and for a neutral variant the probability of that outcome equals its starting frequency, so a single new mutation in a population of size N fixes with probability one over N and, if it does, only after a time on the order of the population size itself. In a population that has grown explosively from a handful of founders in only a few hundred generations, essentially no new mutation has had either the time or the demographic opportunity to sweep to one hundred percent. Strict, population-wide fixation of post-Flood

mutations is therefore negligible, and this holds under any chronology, biblical or evolutionary alike. That is not the quantity a molecular clock counts, and it is not the quantity we count here. What accumulates, and what distinguishes one human mitochondrial genome from another, is an established lineage variant: a mutation that arises on a maternal line is transmitted to every descendant of that line, and is in that sense fixed within the sub-clade it defines, whether or not it ever approaches fixation in the population at large. Under strict neutrality, the population-level substitution rate equals the rate of genuine heritable mutation (Kimura 1968). The composite pedigree-event rate assembled here includes several distinct observational categories: we propose that they can reasonably be treated as estimates of the variation of the substitution rate at which lineage substitutions arise. We treat it as an exploratory pooled high end pedigree-rate summary as stated prior. Over G generations a single lineage is expected to accumulate the product of the rate and G substitutions, distributed as a Poisson variable. Two lineages that last shared a common ancestor T generations ago are expected to differ at twice the rate times T sites, one branch's worth of accumulation on each side. These are the quantities that the observed differences between living people record, and these are what the biblical windows must reproduce. Over G generations a single lineage is expected to accumulate $G / 19.9$ substitutions, distributed as a Poisson variable, and two present-day lineages that separated at the event differ by twice that amount, $2G \div 19.947$ Table 2 projects the expected mitochondrial control-region divergence forward from each candidate event at the pooled high-end mean.

Event and chronology	Generations	Subs per lineage	Expected pairwise divergence
Flood, Masoretic	225	11.3	22.6
Flood, Septuagint	275	13.8	27.6
Creation, Masoretic	300	15.0	30.1
Creation, Septuagint	380	19.1	38.1

Table 2. Expected mitochondrial control-region accumulation at one substitution every 19.947 generations and 20 years per generation. Per-lineage values are $G \div 19.947$; expected pairwise divergence is $2G \div 19.947$.

5.4 Interpreting the Flood and Creation horizons

Read down the right column, two lineages whose common ancestor sits at the Flood are expected to differ by about 22.6 to 27.6 substitutions, and two whose common ancestor sits at Creation by about 30.1 to 38.1. The Creation window at the pooled high-end mean therefore anticipates a deepest human mitochondrial split in the 30-38 substitution range. Within the three-founder structure of Section 5.2 these two figures mark two natural horizons: divergences between lineages that descend from a single Flood woman coalesce at or after the Flood and sit near the lower figure, while divergences between lineages descending from different Flood women coalesce before the Flood, back toward the first woman, and reach the upper figure. The model predicts an expected divergence for lineages approaching the deepest permitted coalescence boundary with a mean of 30.1 to 38.1 substitutions, the split between the most anciently separated founding lineages. Because this is a high-end mean rather than the fastest observed rate, it is expected to reproduce the body of the deep divergences, not the single most extreme pair; that distinction is developed in Sections 5.5 and 5.10.

5.5 Predicted pairwise-divergence ranges

The deepest divergence expected under the two Creation chronologies has a central mean of roughly 30.1 to 38.1 substitutions. The deepest African American pairwise distance in the databases considered here is 32 substitutions (Budowle et al. 1999), and the deepest distance across all eight regional databases is 36. Both representative deep pairs, 32 and 36, fall squarely inside the Creation-timeline prediction. So even the deepest divergence expected under the two Creation chronologies ranges from approximately 30.1 to 38.1 substitutions.

The African American maximum of 32 differences and the all-database maximum of 36 differences both fall within this predicted interval. We want to be precise about how that should be read, because it is central to the logic of this paper. The 19.947 value is a high-end mean of the fastest rates, not the fastest observed rate. The 36-difference pair is the rarest observation in pooled regional databases. An extreme upper-tail observation is matched by the upper-tail of the rate distribution, not by its center. This is why we were just able to capture it using these high rates alone.

The pedigree literature supplies exactly such upper-tail rates: Madrigal et al. (2012), pedigree 10, ran at one substitution every 9 generations across six sequenced transmission events, and pedigree 11 at one every 10 generations, basically the same. Applying an upper-tail rate to an upper-tail observation, and the high-end mean to the representative deep pair, matches like with like rather than forcing a single rate to carry the whole distribution from center to extreme.

The extreme differences are extremely rare, and so are the fastest pedigree rates; the argument here is that both tails exist and line up, not that the mean should stretch to the maximum.

Event and chronology	Per-lineage mean	Deepest pairwise mean
Flood, Masoretic	11.3	22.6
Flood, Septuagint	13.8	27.6
Creation, Masoretic	15.0	30.1
Creation, Septuagint	19.1	38.1

Table 3. Predicted per-lineage and pairwise divergence at the pooled pedigree rate of one substitution every 19.947 generations

The observed maxima falls inside this divergence range. The deepest African American pairwise distance in the databases considered here is 32 substitutions, and the deepest distance across all eight regional databases is 36. Both lie between the Masoretic and Septuagint Creation predictions of 30.1 and 38.1. A recent-origin clock running at the pooled pedigree rate does not merely permit the observed diversity, it places the deepest observed comparisons near the expected divergence for lineages whose common ancestry approaches the upper chronological boundary.

5.6 Analytic pairwise molecular-clock conversion

Treating each observed pairwise distance d as twice the per-lineage accumulation since the common ancestor, the analytic coalescence age is $(d / 2)$ times 19.947 times 20 years. Table 4 applies this to three reference distances: the African American pairwise distance maximum of 32, and the maximum of 36 across all eight databases.

Observed pairwise distance	Substitutions per lineage	Pooled coalescence age (years)
32 (African American maximum)	16	6,383
36 (maximum, all eight databases)	18	7,181

Table 4. Analytic coalescence ages, $(d / 2)$ times 19.947 times 20 years.

The African American maximum of 32 corresponds to about 6,383 years, inside the 7,600-year Septuagint boundary by roughly 1,217 years. The deepest distance of 36 corresponds to about 7,181 years, roughly 419 years within that boundary. The typical and representative deep comparisons both land inside the biblical window at the high-end mean.

5.7 Testing predictions

To test these predictions we need to know what the average pairwise genetic diversity is in the population and what the maximum pairwise diversity is from tip to tip for whether the proposed clock and timescale can accommodate the most divergent observed pair in the dataset. Two studies exist that published maximum diversity for the hypervariable regions, Krings et al. (1999) published modern human vs. modern human: mean 10.9 ± 5.1 , with a max distance of **35 differences** and Budowle et al (1999) reports that 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). The African American within group has the highest within maximum at **32** differences and in all eight datasets combined is **36**.

Figure 3. From Budowle et al 1999 Table 3.

Populations	# Matches / # Comparisons	Freq ($\times 10^{-3}$)	Avg. # Different Nucleotides $\pm$ SD	(Range)
<i>Within group</i>				
Pairwise Comparisons				
African American	24/11026	2.18	14.1 ± 5.8	(0–32)
African (Sierra Leone)	61/6105	9.99	13.1 ± 5.6	(0–31)
US Caucasian	669/182106	3.67	7.9 ± 3.7	(0–30)
Austrian Caucasian	15/5050	2.97	8.4 ± 4.1	(0–26)
French Caucasian	23/5886	3.91	7.2 ± 3.4	(0–19)
Hispanic	36/4851	7.42	12.6 ± 5.7	(0–30)
Asian American	13/1653	7.86	10.9 ± 4.0	(0–24)
Japanese	52/13041	3.99	9.7 ± 3.9	(0–23)
All Eight Databases	1559/969528	1.61	10.6 ± 4.9	(0–36)

5.8 Bayesian pairwise Poisson-clock age analysis

Placing a uniform prior on coalescence time and a Poisson likelihood on the observed distance, the posterior on the age is a Gamma density with shape d plus one and a scale of 19.947 halved and multiplied by 20 years. Its mean is (d + 1) times 19.947 divided by 2, times 20 years. Table 5 gives the posterior means for the two deepest observed distances, and Table 6 reports how much of each unbounded posterior falls within the Masoretic and Septuagint Creation boundaries.

Observed pairwise distance	Unbounded posterior mean (years)
32	6,583
36	7,380

Table 5. Unbounded posterior mean age, (d + 1) times 19.947 divided by two, times 20 years.

Observed distance	Chronological boundary	Posterior mass within boundary
32	Masoretic, 6,000 yr	about 32%
32	Septuagint, 7,600 yr	about 82%
36	Masoretic, 6,000 yr	about 12%
36	Septuagint, 7,600 yr	about 59%

Table 6. Fraction of the unbounded posterior falling within each Creation boundary at q = 19.947.

Applied to the African American deepest pair, the unbounded posterior mean is approximately 6,583 years, with about 82 percent of its mass below 7,600 years. Even the deepest observed distance of 36 has an unbounded posterior mean of approximately 7,380 years, with about 59 percent of its posterior mass falling below the 7,600-year Septuagint boundary.

Thus, under the pooled high-end rate, the Bayesian posterior places the deepest comparisons predominantly below the biblical window of creation. For the deepest $d = 36$ comparison, reducing the assumed generation interval to approximately 16.71 years would place the maximum-likelihood age at approximately 6,000 years.

5.9 Chronologically Truncated Posterior Analysis and Comparison with the Unbounded Maximum-Likelihood Estimate

A complementary analysis conditions on the historical model being tested and asks where within its permitted interval the genetic likelihood places the mitochondrial root. Imposing a historically bounded uniform prior on the pairwise coalescence time, uniform between 0 and 380 generations (7,600 years at 20 years per generation) and zero beyond, and retaining the same Poisson likelihood, gives the truncated posterior summaries in Table 7.

An important distinction emerges when the unbounded likelihood maximum is compared with the boundary. For the representative deep pair, $d = 32$, the maximum-likelihood coalescence age is $(32 \div 2)$ times 19.947 times 20, which is 6,383 years, approximately 1,217 years inside the 7,600-year boundary. For the single deepest pair, $d = 36$, the maximum-likelihood age is $(36 \div 2)$ times 19.947 times 20, which is 7,181 years, roughly 419 years inside the boundary.

The Bayesian calculation in Section 5.8 deliberately used an unbounded uniform prior, so that the genetic likelihood could first be examined without imposing the chronology being tested. A complementary analysis asks a different question: conditional on the historical model being tested, where within its permitted chronological interval does the genetic evidence place the mitochondrial root?

The Septuagint chronology used in this study places the oldest Creation boundary at approximately 7,600 years before present. At the principal generation interval of 20 years, this corresponds to 380 generations:

$$7,600 \text{ years} \div 20 \text{ years per generation} = 380 \text{ generations}$$

We therefore impose a historically bounded uniform prior on pairwise coalescence time, uniformly distributed between 0 and 380 generations. Every interval of equal width between the present and 380 generations ago is assigned equal prior density, while coalescence times older than 380 generations are assigned zero prior density.

This is most appropriately described as a historically bounded, or chronologically truncated, uniform prior, rather than a power prior or commensurate prior. Here, the historical information supplies an explicit upper boundary on the parameter being estimated. The prior density can therefore be written as:

$$\text{Prior probability of } t = 1 \div 380, \text{ for } t \text{ between } 0 \text{ and } 380 \text{ generations}$$

$$\text{Prior probability of } t = 0, \text{ for } t \text{ greater than } 380 \text{ generations}$$

The resulting bounded posterior is not used to estimate the probability that the 7,600-year historical boundary itself is correct. Its purpose is narrower: conditional on the historical model, it shows where within the permitted interval the genetic likelihood concentrates. Evidence concerning the location of the historical boundary is therefore evaluated primarily from the unbounded analysis and the untruncated likelihood maximum presented in Sections 5.8 and 5.10.

The same genetic likelihood used in Section 5.8 is retained. If two mitochondrial lineages last shared a maternal ancestor t generations ago, and q represents the number of generations per pedigree substitution, then the expected number of pairwise differences is: *Expected pairwise differences* = $2t \div q$

At the pooled high-end pedigree mean used throughout this study, $q = 19.947$ generations per event, the probability of observing d pairwise differences is modeled with a Poisson distribution having a mean of: *Poisson mean* = $2t \div 19.947$

Accordingly, the probability of observing d pairwise differences at coalescence time t is modeled by a Poisson distribution with mean:

$$\lambda(t) = 2t \div 19.947$$

No alteration is made to the observed genetic data or to the pedigree-event rate. The only change from Section 5.8 is that the posterior distribution is restricted to the historical interval from the present to 7,600 years ago. Under an unbounded uniform prior, the posterior distribution for t is a Gamma distribution with a shape parameter of $d + 1$ and a scale parameter of $q \div 2$. For the historically bounded model, this same posterior is retained between 0 and 380 generations and truncated above 380 generations. The resulting posterior can therefore be described as proportional to t raised to the power d , multiplied by e raised to the power of negative $2t$ divided by q , for values of t between 0 and 380 generations.

The unbounded posterior above allows arbitrarily old ages with vanishing probability. A young-earth model instead treats the biblical upper creation date of 7,600 years as a hard upper boundary and truncates the posterior there. Table 7 reports the mode, median, mean, and 95 percent credible interval of the truncated posterior for the two deepest observed distances, together with the conditional mass that falls between the Masoretic and Septuagint boundaries.

d	Mode	Median	Mean	95% credible interval	Mass 6,000 to 7,600
32	6,383	6,256	6,192	4,456 to 7,506	60.7%
36	7,181	6,687	6,578	4,993 to 7,553	79.3%

Table 7. *Historically bounded (truncated at 7,600 years) posterior summaries at one substitution every 19.947 generations. At this rate, the unbounded likelihood maxima for both $d=32$ and $d=36$ fall within the 7,600-year boundary, so neither truncated posterior mode is forced against the upper boundary.*

For the representative deep distance, $d=32$, the posterior mode coincides with the unbounded maximum-likelihood age at 6,383 years, approximately 1,217 years inside the 7,600-year boundary, because the most probable number of generations is $d/2$ times the rate: $(32 \div 2) \times 19.947$, which is 319.152 generations, or 6,383 years at 20 years per generation.

For the single deepest distance, $d = 36$, the unbounded maximum-likelihood age is $(36 \div 2)$ times 19.947, which is 359 generations, or approximately 7,181 years, which lies about 419 years inside the 7,600-year boundary. Because this maximum already falls within the historically permitted interval, truncation does not force the posterior mode against the boundary; the bounded mode remains at approximately 7,181 years. Truncation instead removes the older tail of the posterior, shifting the bounded median and mean downward while leaving the location of the likelihood maximum unchanged.

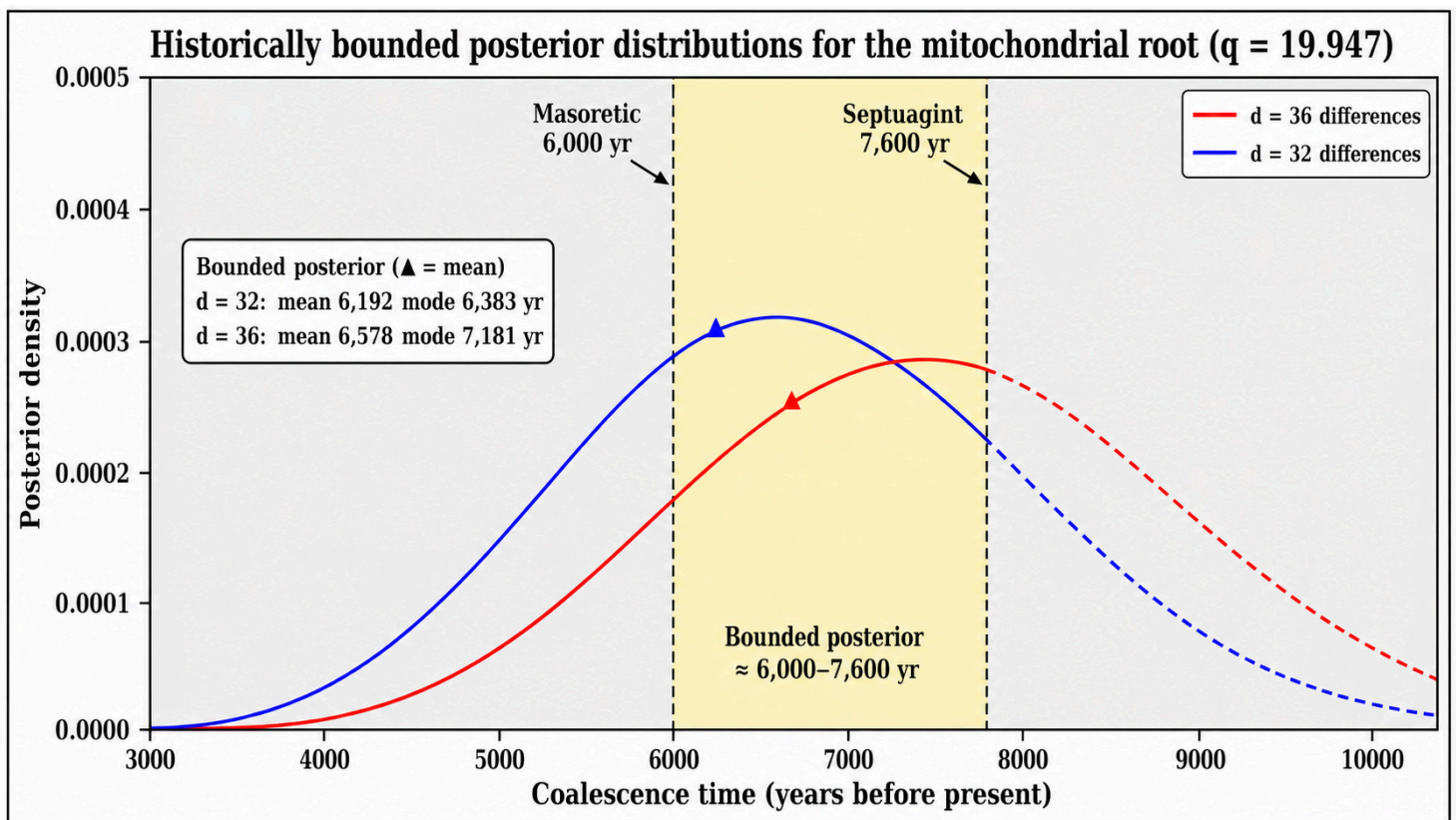


Figure 4. Historically bounded posterior distributions for the mitochondrial root at $q = 19.947$. The solid curve for each comparison is the posterior retained within the 7,600-year upper biblical boundary; the dashed continuation is the tail removed by the boundary. The shaded band marks the 6,000 to 7,600-year Creation interval, and each triangle marks the bounded posterior mean.

5.10 Posterior Mean Versus Likelihood Maximum

Because the historically bounded posterior cannot independently test the upper boundary imposed upon it, the primary chronological comparison is made using the untruncated likelihood maximum. An important distinction emerges when the unbounded Bayesian result is compared with the maximum-likelihood estimate. For an observed pairwise difference count of 36 and a pedigree substitution interval of one substitution every 19.947 generations, the maximum-likelihood coalescence time is $(36/2) \times 19.947 \times 20$, which is $18 \times 19.947 \times 20$, or approximately 7,181 years. The unbounded posterior mean is approximately 7,380 years, whereas the maximum-likelihood estimate is approximately 7,181 years. The difference arises because the unbounded Bayesian posterior is asymmetric and possesses a right-hand tail extending toward older ages. That tail pulls the posterior mean upward by approximately 199 years, to about 7,380 years, even though the probability density itself reaches its maximum at approximately 7,181 years.

In simpler terms, the two values answer slightly different questions. The maximum-likelihood estimate asks which single age provides the best fit to the observed 36 differences, yielding approximately 7,181 years. The posterior mean instead averages across the entire range of possible ages, weighting each according to its posterior probability. Although ages above 7,181 years become progressively less probable, the older side of the distribution extends farther than the younger side. Those individually less probable older ages collectively pull the average upward to approximately 7,380 years. The 199-year difference therefore does not represent disagreement between two genetic clocks or two datasets; it is a mathematical consequence of summarizing the same asymmetric probability distribution in two different ways. Both measures place the inferred coalescence within essentially the same recent chronological interval.

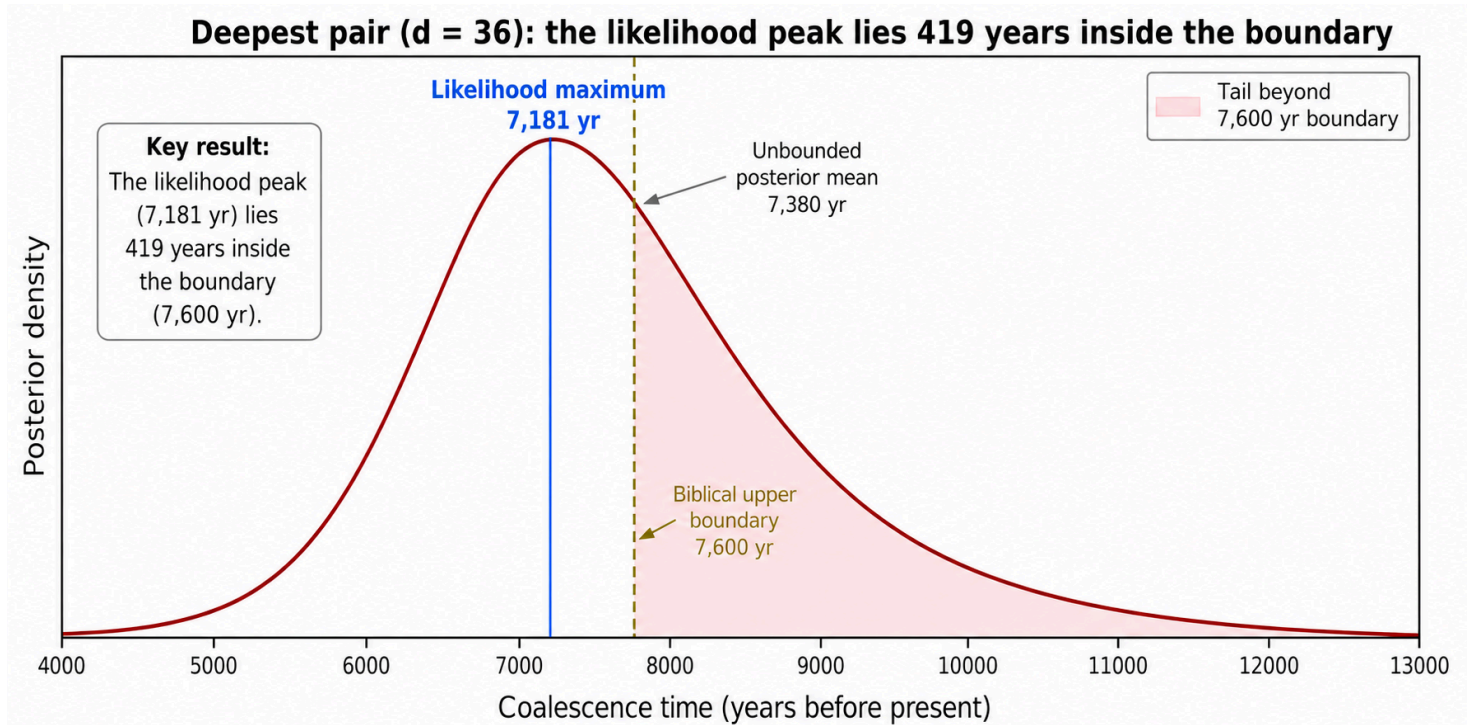


Figure 5. Unbounded posterior for the deepest pair ($d = 36$). At a pedigree-event interval of one substitution every 19.947 generations, the likelihood maximum occurs at approximately 7,181 years, about 419 years inside the 7,600-year biblical upper boundary, while the asymmetric right tail raises the unbounded posterior mean to approximately 7,380 years.

Two summaries of the deepest-pair posterior differ, and the distinction matters for how the result is stated. For an observed pairwise difference count of 36 and a pedigree event interval of one substitution every 19.947 generations, the maximum-likelihood age, the peak of the density, is approximately 7,181 years. The unbounded posterior mean is approximately 7,380 years, about 199 years older than the likelihood maximum because the uniform-prior posterior is asymmetric and possesses a right-hand tail extending toward older ages. The genetic likelihood maximum falls approximately 419 years inside the 7,600-year boundary, while even the unbounded posterior mean remains approximately 220 years inside that boundary.

6. Somatic and recurrent mutations, male-line exclusion, and the saturation correction

It is true that mutations can turn over in the same place, we refer to them as saturation, parallel mutations or recurrent mutations. Saturation accumulates with the number of generations: at a few hundred generations the probability of a second, masking hit at the same site is small, whereas at the tens of thousands of generations implied by a deep time is large. We can agree to this, but our calculations run over just over roughly a few hundred generations max, the horizon on which saturation is minor. The timeframe tested in the present model spans only approximately 300 to 380 generations.

It therefore does not require the pedigree rate to remain constant across the tens of thousands of generations assumed in conventional phylogenetic chronologies. Pedigree and phylogenetic rates are treated here as separate calibrations belonging to different temporal models, and no phylogenetic rate is imported into the recent-origin calculations. Long-term time-dependent rate decay may be relevant to models extending human mitochondrial history over hundreds of thousands of years, but it is not a correction to a model confined to only a few hundred generations. For the present analysis, the relevant empirical question is whether the observed pedigree-event rate remains a reasonable approximation across approximately 300 to 380 generations, not whether it persists indefinitely across deep evolutionary time.

We claim that on a recent-origin model the saturation correction is extremely small and therefore trivial and makes no difference in the clock's rate in our timeline. The dispute between the two frameworks is ultimately about which timescale is correct, and it is the phylogenetic model that cannot reconcile the two rates, so the problem is theirs solely, for us it is no problem at all. This section is merely an overview of the topic. The reason we creationists favor the pedigree rates in the D-loop is that it is not just because they are observable, testable, repeatable and land on the Biblical timeline. No, those were just what caught our attention.

The fact is, they are the only rates lining up with known historical dates: such as documented bottlenecks and breeding events in other species, including whales (*Marcos Suarez Menendez et al 2023*), Holstein cattle (*Koehler et al. 1991*), cats (*Jennifer D. Kurushima 2012*) Horses (*Hill et al, 20002, Jansen et al. 2002*), and humans (*Helgason et al. 2006*) they always recover dates consistent with the recorded known events or timeframe. The Holstein paper concerns rapid heteroplasmy segregation. The cat paper concerns breed and population relationships corroborating the pedigree clock independently recovering a historical event. The 2002 horse paper by Jansen et al., explicitly interprets the genetic results in relation to archaeological evidence that is again validated by the pedigree clock.

While phylogenetic rates may make testable predictions, they only do so either by retrodictions (*after the evidence is in*) or anchoring on to dates they need to reach, obtained from radiometric dating, then calibrating to reach that date. A far cry from the blind pedigree rates, that, on their own, land on known historical events with no need for calibration. If the roles were reversed and creationists were using phylogenetic methods, we would get flagged for using poor and biased methodology. Essentially retrofitting the data to match our pre-conceived story. They are essentially saying they trust the story over the evidence and that the radiometric dates are more valid than observed genetic evidence regardless of what the results are. Therefore we argue in Sections 6, 7 and 8 that the recent-origin biblical model of ancestry fits the data better than the current evolutionary model of explaining the diversity of life on earth.

To put evolution to the test, 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) was directly performed to answer this question. Mentioned at the start of this paper, they looked at 321 maternal transmissions, across 48 lineages covering the full D-loop region which includes both HVR1 + HVR2 and detected 13 new mutations (*11 substitutions + 1 possible deletion + 1 deletion*), affecting 12 distinct positions in 321 transmissions. That gives a raw familial rate around 1 mutation every 24.7 generations (*similar to Howell et al 1996*), a very fast rate yet again. With a pedigree depth average of 6 generations in 26 families, Santos explicitly explored what happens when you compare the raw data vs. filter and exclude certain mutational variants. While the study covered many topics, we are going to focus on recurrent mutations first, then somatic then male bias. The only position in the entire dataset that mutated twice was 16189, a genuine site that arose independently in two families, so the honest recurrence fraction is 1 of 12 positions, 8.3 percent. But that number cannot be extrapolated.

Saturation scales with divergence, roughly as rate times time relative to the number of hittable sites, and over a pedigree depth of 5 to 6 generations it is effectively zero and even over just a few hundred generations it makes essentially no difference which we will get into soon. The single recurrence reflects one length unstable hotspot, not a population wide constant as well. The correct tool is a finite sites or gamma distributed rate correction that grows nonlinearly with divergence, and it would not sit at 8.3 percent. So the raw rate stands where the data put it, 1 substitution every 24.7 to 29 generations depending on whether you count all 13 events or the 11 substitutions, the same fast neighborhood as Parsons. And Santos never tried to close the pedigree to phylogenetic gap with saturation or recurrent hits in the first place. They explicitly rejected the hypervariable site saturation explanation, noting that family study mutations do not fall preferentially on hot spot sites. The correct tool is a finite-sites or gamma-distributed-rate correction, which grows nonlinearly with divergence and would not remain at 8.3%. So the rate would still remain at around 1 substitution every 24.7 to 29 generations depending on the raw rate you trust more. 24.7 counts all 13 events and 29 counts the 11 substitutions.

Here is the question for the evolutionary side. How saturation compounds with assumed time (slowdown = how much slower the observable, or effective, rate is than the raw pedigree rate). We ran under a uniform-sites JC69 baseline finite-sites correction, since the control region is known to concentrate change in a few fast HVR sites: The next figure shows over the 6,000 year timeline how much of a slowdown would be expected based on this uniform Jukes Cantor correction heterogeneity correction:

Finite-sites slowdown for tip-to-tip (pairwise) divergence			
610-bp HVR-I/HVR-II window; rate = 1 substitution per 19.947 generations per lineage			
Assumed MRCA divergence time	Generations to each lineage*	Expected pairwise subs/site†	JC69 (uniform) % loss of raw subs (% observable)
6,000 yr	300	0.0493	3.2% (96.8%)
* Generations from the MRCA to each descendant lineage (20 years per generation).			
† Expected pairwise substitutions per site across the two lineages: subs/site = 2 × generations / (19.947 generations per substitution) / 610 bp.			
Note: Values show the percent of accumulated substitutions that are hidden by recurrent/parallel substitutions under finite-sites models. Parentheses indicate percent of raw substitutions expected to remain observable.			

Figure 6. Saturation slowdown over time under the pooled pedigree rate. The table shows the amount of the raw substitution signal expected to be hidden by recurrent and parallel mutations when the combined 610-bp HVR-I/HVR-II window is modeled at one substitution every 19.947 generations per lineage. JC69 represents a baseline uniform-sites model. At 6,000 years, corresponding to 300 generations at 20 years per generation, the JC69 model conceals only about 3.2% of accumulated substitutions, leaving about 96.8% still observable. Saturation is strongly time dependent, but over the few hundred generations relevant to the recent-origin model, the ordinary finite-sites correction remains small and makes little difference over the timeline.

6.1 Still insufficient to bridge the pedigree–phylogenetic gap

Even when the pedigree rate is projected forward for 200,000 years, the uniform-sites JC69 correction does not erase enough substitutions to reduce the expected diversity to the level examined here. At one substitution every 19.947 generations per lineage, 200,000 years corresponds to 10,000 generations from the MRCA to each descendant lineage and approximately 1,003 raw pairwise substitutions across the 610-bp HVR window. Under the JC69 uniform-sites finite-sites model, approximately 406 differences remain observable. By comparison, the maximum pairwise difference published by Budowle *et al* is used in the present analysis at 36.

Expressed as a slowdown factor, the 200,000-year JC69 correction amounts to approximately 2.47-fold. To compress the roughly 1,003 raw substitutions predicted by the pedigree-rate projection all the way down to 36 would instead require a slowdown of approximately 27.9-fold, meaning about 96.4% of accumulated substitutions would have to be hidden from observation. The uniform-sites JC69 model conceals approximately 59.5%, rather than 96.4%. Under this baseline model, saturation therefore contributes substantially to rate slowdown over deep timescales but is quantitatively insufficient by itself to reduce the pedigree-rate projection to 36 differences. The contrast is considerably smaller over the timescale actually tested by the recent-origin model. Between 300 and 380 generations, the uniform JC69 calculation hides only approximately 3.2% to 4.1% of accumulated substitutions, leaving approximately 96% to 97% observable.

Thus, over only a few hundred generations, recurrent mutation can only modestly modify the expected count and does not fundamentally reshape either the results or the pedigree signal. A second way to demonstrate this is to begin with the divergence actually observed rather than with an assumed age. At approximately 2% observed sequence divergence, the ordinary JC69 multiple-hit correction increases the inferred number of substitutions by only about 1.36%. Under the pooled rate of one substitution every 19.947 generations per lineage, a naive 2% pairwise divergence across the 610-bp HVR-I/HVR-II window corresponds to approximately 2,434 years. Applying the JC69 correction increases this estimate to only about 2,467 years, a change of roughly 33 years.

This illustrates an important distinction. The large saturation percentages obtained when the pedigree rate is projected across 200,000 years arise only after extrapolating that rapid rate across 10,000 generations per lineage and allowing a very large number of substitutions to accumulate. They are not corrections demanded by the comparatively small amount of sequence divergence actually observed among living humans. Under the recent-origin model examined here, the observed divergence therefore requires at most, only a minor finite-sites correction.

The multiple-hit correction is controlled primarily by how much sequence divergence has accumulated, not simply by assigning an older chronological age to the sequences. Small observed divergence provides relatively little opportunity for recurrent substitutions to have hidden large numbers of previous changes. Conversely, if the pedigree rate of one substitution every 19.947 generations were projected continuously across 200,000 years, the predicted raw accumulation would become sufficiently large for saturation to be substantial. Even under that deep-time projection, however, the uniform-sites JC69 correction does not conceal enough substitutions to reduce the pedigree-rate expectation to the observed level examined in this study. Santos et al. (2005) likewise did not reconcile the discrepancy between pedigree-derived and phylogenetic rates through recurrent-mutation saturation alone.

Their analysis considered recurrent sites but placed the major reduction elsewhere: in distinguishing presumed germline from presumed somatic events, excluding mutations carried only by males when estimating long-term maternal-line contribution, and applying a probability that an intraindividual heteroplasmy would eventually reach fixation. Their germline D-loop estimate was reduced from 0.7684 to 0.1675 substitutions per site per Myr after these additional corrections, while their HVR-I/HVR-II corrected estimate was 0.2415 per site per Myr. These reductions therefore arose primarily from biological classification and fixation assumptions rather than from finite-sites saturation itself.

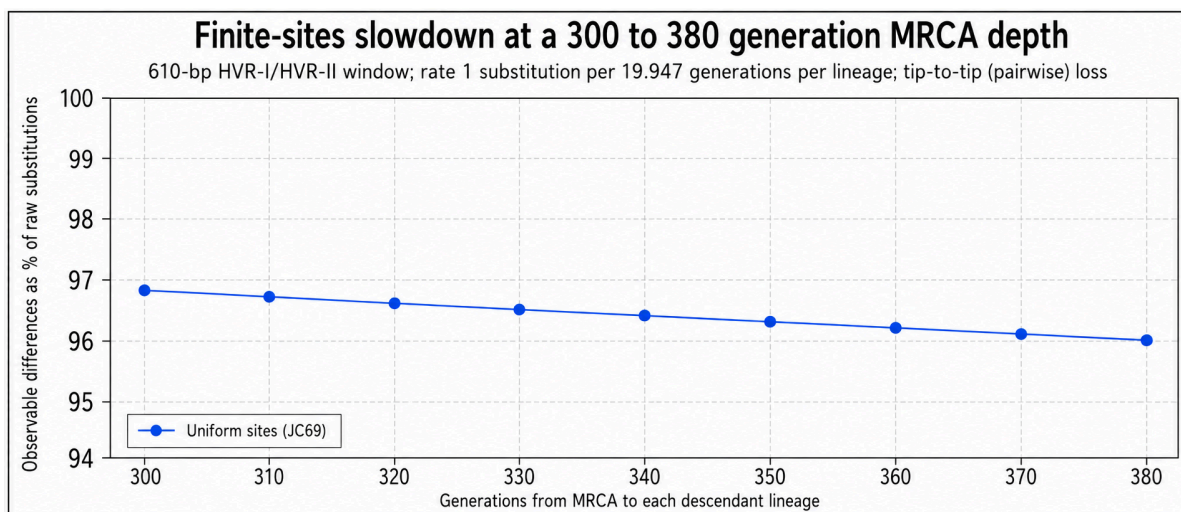


Figure 7: Finite-sites slowdown for the tip-to-tip (pairwise) divergence between two lineages whose common ancestor lies 300 to 380 generations back, across a 610-bp HVR-I/HVR-II window at one substitution every 19.947 generations per lineage. Under a uniform-sites JC69 model, recurrent and parallel hits conceal approximately 3.22 to 4.05 percent of the accumulated pairwise substitution signal, leaving approximately 96.78 to 95.95 percent observable.

Recurrent and parallel substitutions produce a measurable but limited finite-sites slowdown over the timescale examined here. In simulations of a 610-bp HVR-I/HVR-II window at one substitution every 19.947 generations per lineage, approximately 95.9 to 96.8 percent of the raw substitution signal remains observable in the tip-to-tip pairwise divergence between two lineages whose common ancestor lies 300 to 380 generations back under a uniform-sites JC69 model. At 300 generations, approximately 3.2 percent of accumulated substitutions are concealed by recurrent or parallel hits, while at the deepest 380-generation comparison the corresponding loss rises only to approximately 4.1 percent.

The important point is that 300–380 generations is simply not long enough, under the uniform JC69 finite-sites model, for repeated mutations at the same sites to erase most of the genetic signal. Even at the deepest 380-generation comparison, only about 4.05 percent of the raw substitution signal is hidden by recurrent hits, meaning approximately 95.95 percent remains observable. Saturation can therefore make the clock run slightly slower over a few hundred generations, but the effect is quantitatively small: at most about 4 percent of the accumulated signal is concealed across the timescale examined here. Such a correction is far too small to substantially alter the chronology produced by a pedigree rate of approximately one substitution every 19.947 generations per lineage, and it does not transform that pedigree rate into anything remotely approaching the much slower long-term phylogenetic rates. For a model spanning only about 300–380 generations, the correction changes the result negligibly rather than fundamentally. That is the entire point we are making, applying evolutionary slow down mechanisms that make no difference in our model, they are strictly mechanisms evolution needs to apply to their own model to help reconcile the observed paradox.

6.2 Partitioned (among-site rate variation) sensitivity

The uniform JC69 model treats every site as equally mutable, whereas the mitochondrial control region exhibits substantial among-site rate heterogeneity. A gamma-distributed-rate extension therefore provides a stricter sensitivity test. Re-running the 610-base-pair HVR-I/HVR-II window at one substitution every 19.947 generations per lineage under a gamma-rate JC69 model increases the multiple-hit correction. At 380 generations, approximately 95.95% of the raw substitution signal remains observable under uniform JC69, compared with approximately 89.02% under moderate rate heterogeneity (gamma shape $\alpha \approx 0.5$) and approximately 80.76% under strong heterogeneity ($\alpha \approx 0.2$). These correspond to losses of approximately 4.05%, 10.98%, and 19.24%, respectively. Even under the strong-heterogeneity model, therefore, about four fifths of the accumulated raw signal remains observable. This is still far short of the approximately 96.4% loss that would be required to compress the 200,000-year pedigree-rate projection from roughly 1,003 raw substitutions to the 36-difference benchmark examined here. Allowing substantial among-site rate variation therefore increases the finite-sites correction over a few hundred generations, but it does not alter the central result: saturation remains quantitatively insufficient to erase most of the pedigree signal over the 300–380-generation interval.

The distinction between germline and somatic events was also not experimentally certain in every case. Santos et al. 2005 reduced the original eleven substitutions to six presumed germline events by classifying five singleton changes as potentially somatic, while acknowledging that such heteroplasms could alternatively have originated in the germline during oogenesis. Their somatic call did something narrower but more consequential to the arithmetic: when they reclassified 5 of the 11 detected substitutions as somatic, which alone cut the standardized rate from 1.4088 down to 0.7684 mutations per site per Myr, before any other correction, they literally cut the mutation rate almost in half (a 45.5% reduction). They were candid that the line between germline and early-somatic events cannot be drawn with certainty and that their corrected rate is therefore a lower bound, stating; *“...in these families the reported heteroplasmy **may** have resulted from somatic mutations that occurred in the individuals themselves, or alternatively, it may have resulted from germ line mutations that occurred in the oocyte from which the heteroplasmic individuals developed.”* In other words, Santos admits the mutations they set aside as somatic could very well be germline events that simply weren't caught in the mother, a real possibility given that direct Sanger sequencing does not resolve low level heteroplasmy. Again, they admitted they would need more generations to determine the true rate. Reverse that one uncertain call and the germinal rate roughly doubles back toward the raw figure we find in the majority of other pedigree studies. Which is what they later admit is probably the case and why they published the raw rate as well which kept in those mutations. They further noted that a rigorous theoretical estimate of the probability that a heteroplasmic mutation ultimately reaches fixation was *“currently impractical”* and that direct empirical resolution would require observations across additional generations. One in which their study could not supply. Their analysis therefore demonstrates an important point for the present discussion: somatic mutation count was an assumption and finite-sites saturation alone does not produce the required slowdown; most of the reconciliation must come from additional assumptions about mutation origin, lineage survival, and fixation.

Since heteroplasmies can turn into fixed substitutions, observing and counting them and accounting for them is an important aspect of any rate, that is why they have detection thresholds lower nowadays. This is why when we see studies like Zaidi et al., (2019) at 1%, Wei et al., (2019) at 1%, and Peng et al., (2025) at 6% who all set a lower detection filter, observed many more heteroplasmic mutations that they thought might drift away rapidly, but they rather drifted rapidly to fixation and were true mutations. Unsurprising to us, those studies also obtained a similar rate to *Parsons* even though *Zaidi* and *Peng* were deeper pedigrees. *Santos* further cautions that their final published rate approach is conservative and that the true germline mutation rate may be somewhat higher than the corrected estimate and closer to the raw rate. Their “*corrected rate*” is a lower bound, not a definitive rate. This is why they still kept their raw observed rate and it became their upper bound. Again 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 conventional evolutionary paradigm driving the conclusions. They explicitly stated that their filtering strategy intentionally erred on the side of undercounting, explaining “The reported heteroplasmy may have resulted from somatic mutations ... or alternatively, it may have resulted from germ line mutations.” In the supplemental material we can read again regarding the three families described above: “*They must result from somatic mutations ... or alternatively, it could result from germline mutations that occurred in the oocyte.*” The supplement specifically applies this uncertainty to the CAR_I, CAR_II, and PA_I mutations stating almost the same thing. “*May represent a somatic mutation ... or a germline mutation that occurred in the oocyte that gave rise to the individual.*”

Again, they admitted that their rate after filtering could most likely be off, and if we look at the observed rate it aligns perfectly yet again with *Parsons* and many others. *Santos et al.* applied a conservative evidentiary rule; their six-event calculation therefore represents a conservative count of demonstrated germline events; the actual germline rate could be higher, although the study could not determine by how much.

In Howell's pooled pedigree data study they considered this and reported on it, we read: “*Sigurðardóttir 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 criterion 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. (1997), which analyzed small pedigrees, the newly arising mutations were detected in multiple family members and thus cannot be somatic. Somatic mtDNA variants do occur (table 3; see also Howell et al. 1996), but they will not limit pedigree analyses if the appropriate inclusion criteria are used.*” (Emphasis added). So with somatic mutations not being the factor that they need to slow the rate down, they need to reach for other factors. Those factors include purifying selection and background selection mechanisms which we investigate in section 7.

Pedigree studies that are reporting results obtained from fixed substitutions cannot have somatic mutations as an argument against them since somatic mutations are never inherited from the parent. The mistake geneticists can make with somatic mutations come from heteroplasmy, one cannot distinguish what is somatic and what is not depending on the frequency of the mutation. This is why some studies directly count only fixed homoplastic results since they do not include possible somatic mutations in their results. If they truly were counting somatic mutations by accident all the time in these single generation pedigrees, then the rate would slow way down in deeper pedigree studies such as *Zaidi et al.*, (2019), *Mumm et al.*, (1997) *Forster et al.*, (2002) and *Howell et al.*, (1996) who all followed multiple transmission deep pedigrees, yet obtained results that ranged from one substitution every 19.2 – 34.6 generations, all right next to *Parsons et al.* So in closing, *Santos* showed that depending on how transitions are classified (germline vs. somatic/artifactual), the rate can vary by almost an order of magnitude. *Santos* in the end admitted that the mutations that were filtered out may have been true mutations and that there was no real way to know unless their pedigree was deeper. We state that other pedigrees do exist that are deeper and the rate is near their raw rate, not their filtered rate. Which means their assumptions were wrong.

A further correction that several pedigree studies apply, and that we have so far only touched on, concerns the sex of the individual in whom a new substitution is inherited and detected. A germline mutation arising in a mother's oocyte pool is transmitted to sons and daughters alike, so males acquire new control-region substitutions at the same rate females do, and Parsons et al. (1997) and Santos et al. (2005) both recorded this. Santos went one step further and reasoned that because a male cannot pass his mitochondrial DNA to the next generation, any germline mutation carried only by a male is an evolutionary dead end and should be removed when estimating a long-term substitution rate. On that basis, Santos discarded two of the six germline mutations they detected, the variants in individuals HR_I IV2 and ML III2, which lowered their standardized germinal rate from 0.7684 to 0.5123 mutations per site per Myr, a reduction of about one third before the separate intraindividual fixation step was even applied. So if Santos did not have an evolutionary mindset and was not trying to close some imaginary phylogenetic gap, her and her team would not have considered many if any variants to be somatic, nor would they be filtering out males to slow the rate down. This male bias approach alone dropped the rate from 11 down to 9 out of 321 transmissions which is a rate of one substitution every 36 generations. Santos was explicit that this gender correction is what helps the corrected figure to land in the upper end of the phylogenetic range, which tells us plainly what the correction was reaching to obtain, evolutionary results to bridge the gap between pedigree rates and phylogenetic rate which has been in contention from its discovery to this day. A reader may ask why later pedigree studies do not adopt the male-exclusion correction that Santos et al. (2005) applied, in which mutations inherited by sons are removed on the grounds that a male cannot transmit his mitochondrial DNA onward. Clearly this would help them slow the rate down to help bridge the gap a little closer to the evolutionary phylogenetic rate. The reason is not oversight but estimand. A new control-region mutation arises in the mother's germline, and because the sex of a child is fixed by the sperm independently of that mutation, sons and daughters inherit newly arising maternal mutations at exactly the same rate; the mutation process itself is sex-blind. Removing male-carried variants therefore does not correct the per-transmission mutation rate at all. It answers a different question, namely how fast the population's maternal gene pool turns over, a quantity in which copies landing in males genuinely drop out. For a coalescence calculation the correction is not merely unnecessary but circular. The pairwise differences we read are drawn from living people, and every living mitochondrial genome reached the present through an unbroken chain of mother-to-daughter transmissions; conditioning on a surviving female line has already excluded the sons. To subtract male-carried mutations a second time, by hand, is to remove the dead-end lineages twice, once in the sampling and once in the arithmetic, which pushes the estimate below the true rate rather than toward it. Under the neutrality that holds in the control region since the region does not code for proteins and mutations that hit are neutral, the long-term substitution rate already equals the full germline mutation rate, because the fixation probability of each variant exactly offsets the number arising, an identity that integrates over every variant destined to be lost, including those carried by males. The male-exclusion step, layered on top of that identity, does not recover the substitution rate; it depresses it. This is why the raw, sex-inclusive per-transmission rate is the appropriate estimator for a neutral coalescence clock, and why no prior studies ever incorporated Santos' sex bias model. Our coalescence calculation asks a different question. It reads the pairwise differences between the mitochondrial genomes of living people, and every living mitochondrial genome reached the present through an unbroken chain of mother to daughter transmissions. Conditioning on such a surviving female line already removes the sons: along any lineage that survives to be sampled, every transmission is to a daughter who carried the line forward, and new germline mutations accumulate along it at the full per-transmission rate, since the sex of a child is fixed by the sperm and is independent of whatever mutations arose in the mother's germline. To then subtract the male-carried mutations a second time, as Santos does, is to remove the dead-end lineages twice, once by conditioning on survival and once by hand. The point is sharper still under the neutrality we argue holds in the control region and the hypervariable sites. There the long-term substitution rate already equals the full germline mutation rate, because the fixation probability of each new variant exactly offsets the number arising (Kimura 1983), and that identity integrates over every variant destined to be lost, including those carried by males. For a neutral coalescence clock the raw germline rate is the correct estimator, and Santos's male-corrected figure is an underestimate. Their germline-only estimate was 0.7684 mutations/site/Myr, which they call one of the highest values reported so far and compared it to Parsons et al. directly, and their pedigrees are far deeper than which averaged 6 transmissions deep while Parsons et al. were mostly dyad and some triads.

The substantive reconciliation of pedigree and phylogenetic rates must instead be sought in the fixation dynamics of heteroplasmy or other means, as Santos et al. themselves concluded, rather than in saturation of the mutational process or male bias. They sought after somatic mutations to slow their own rate down, but other studies that accounted for somatic mutations found this argument inadequate and no longer on the table for reconciling the gap. Any complete reconciliation must therefore rely primarily on additional mechanisms outside of selection, saturation, male bias or somatic mutations. Our study here has shown that fixation dynamics of heteroplasmy are compatible with our model and that neither selection, male bias, recurring parallel mutations or saturation solve the evolutionary paradox and have no bearing on our model whatsoever.

7. Background selection and purifying selection and time dependency objections

This is essentially the "linked selection" (background selection/hitchhiking) hypothesis: since mtDNA doesn't recombine, purifying selection removing deleterious coding-region mutations should drag down diversity everywhere on the molecule, including the control region. This makes a good argument on the surface but this is an old idea and has already been dealt with, (Kivisild et al., 2006) proposed exactly this. If that mechanism were sufficient, the low control region diversity would carry no implication about the age of the common ancestor, since it could be produced by selection alone on any timescale. The objection therefore has to be met on its own terms, and when it is tested quantitatively rather than asserted, it does not close. The control region is non coding, so direct purifying selection cannot act on it; any selective reduction there must be inherited by linkage from the coding target. Woodhams (2006) worked out what that requires. Fitting a purifying selection model to the observed decay of rate estimates over time, **excessively high effective population sizes are required**. The coding target on the mitochondrial genome is simply too small for background selection from it to account for the reduction, and Woodhams concluded that this casts doubt on whether deleterious mutations can explain the apparent rate acceleration at all. Nor does the point rest on that paper alone. A few years later Henn et al. (2009), examining human mtDNA specifically, reached the same judgment on the selection component. Citing Woodhams, they noted that the effective population sizes purifying selection alone would demand, **are not realistic**, and turned to other factors to try and account for the discrepancy. Between an analytic treatment of the general case and a direct analysis of the human data, the shared finding is the same: purifying selection on the coding region, operating through background selection, is quantitatively **insufficient** by itself to produce the linked reduction observed in the control region. These prior pedigree studies have already considered this topic long ago and ruled it out, we read *"Sigurdardottir et al. (2000) conclude that **selection is unlikely to be a major factor that underlies the difference between phylogenetic and pedigree divergence rates**, which is not the same thing as saying that selection does not act on mtDNA."* This is further elaborated on by Howell et al. in 2003 regarding the pedigree/phylogenetic gap being real and not a selection artifact of the region: their line that *"there is a real discrepancy"* between the two rates, and their statement that *"**selection has not preferentially distorted the pedigree mutation spectrum relative to the phylogenetic one.**"* This is telling, this is years later and still agreeing with Sigurðardóttir in 2000. As a matter of fact he even quotes Sigurðardóttir in his 2003 paper, stating: *"**selection isn't what's driving the control-region signal.**"* Soares et al. (2009) regarding purifying-selection comparison states: *"The virtually linear relation between the accumulation of synonymous mutations and control-region mutations suggests that **the control region cannot be greatly affected by purifying selection**"* 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). However, there are a number of mutational hot spots in the **control region, ~3% of the total sites, that have a rate of substitution greater than the neutral rate, at some sites by more than an order of magnitude.*** Those "sites" he is referring to were not slower sites by any means, those 3% were faster than expected, so the region is not just effectively neutral, it's faster than neutral. Not slower. The opposite of what they need to slow a rate down over time. Then we get the biggest confession of all: *"As discussed by Sigurdardottir et al. (2000), **it is, rather, a phylogenetic rate estimate that may be biased.**"* Regarding Tennesen et al., this is arguably even more interesting. They concluded that recent rapid population expansion produced an enormous abundance of **young, rare coding variants**, including potentially harmful ones. Each individual carried roughly 13,595 SNVs in the regions examined; about **2.3% were predicted to affect protein**

function, involving roughly 313 genes per individual, and approximately 95.7% of predicted functionally important variants were rare. They attributed the excess of rare functional variation to the combination of recent explosive population growth and weak purifying selection. This tells us that even early on in recent times of human history selection was weak. Now apply that to the distant past with even smaller populations, the problem only compounds. Harpending et al. 1998 directly states what they believe; "Several nuclear loci are informative about our ancestral population size during nearly the whole Pleistocene. They indicate **a small effective size, on the order of 10,000 breeding individuals, throughout this time period.**" (PNAS 95:1961). They are referring to the whole-species, not lineage-specific, not post-out-of-Africa either, and it explicitly covers "the whole Pleistocene era" which covers the entire couple hundreds of thousands of years of our supposed existence. Then there are times where the numbers drop even more. Hu et al. 2023 (Science) states; "the FitCoal analysis inferred an ancestral population fall to roughly 1,280 breeding individuals with the low point lasting about 117,000 years, around 930,000 to 813,000 years ago." These numbers show us that purifying selection in our past is off the table regarding these D-loop region rates and differences and why Sigurdardottir in his 2003 paper, stated: "selection isn't what's driving the control-region signal." Charlesworth 2009; explicitly connects long-term human N_e with weaker selection. "Species with historically low effective population sizes, **such as humans, show evidence for...reduced effectiveness of selection.**" Charlesworth gives a human N_e of approximately 10,000 – 20,000 and attributes it to humanity's long history of comparatively small numbers followed by only a recent expansion. In the American Journal of Human Genetics, Dhindsa et al. (2020); talks about small human population sizes of the past that impairs purifying selection; "Because of the small effective population size of human beings, natural selection is less effective in purging weakly deleterious mutations." Haerty and Ponting (2013); drift is described as the principal force in the past, not selection. "**Genetic drift appears to be the main driving force in the evolution of intergenic lncRNAs, at least in humans.**" They attributed this specifically to small human effective population size as well. Henn et al. 2016; invokes serial founder effects during the Homo sapiens in their paper, stating; "Most predicted deleterious mutations have evolved as if they were neutral during the expansion out of Africa." The authors associated this with repeated founder events, reduced effective population sizes, and stronger genetic drift. This same bottleneck rescue device can be found throughout the literature, so that begs the question. If the effective population size is too small, selection cannot act upon the mitochondria to alter it. Their own model ironically does not allow for selection to be used as a rescue device. This rolls over to Archaic hominins as well: Green et al. (2008); reduced selection in Neanderthal mtDNA. "Purifying selection in the Neanderthal mtDNA was reduced compared with other primate lineages." Their effective population size was even far below the homo-sapien population. Petr et al. (2019); notes the same, small Neanderthal N_e decreased purifying-selection efficacy. "Low effective population sizes...in Neandertals led to decreased efficacy of purifying selection and the accumulation of weakly deleterious variants." None of this is undercut by critics pointing to studies documenting background selection in modern human large populations either. Of course selection is detectable today: the effective population size is enormous, purifying selection has an immense pool of variation to act on, and it is operating as efficiently as it ever has. But that observation answers a different question than the one at hand. The relevant population is not the present one; it is the population that existed at whatever point in the past produced the low control-region diversity the phylogenetic rate depends on. And it is precisely at that point that the selection explanation collapses under its own arithmetic. Woodhams and Henn et al. did not fail to find selection today, they found that the amount of it required to explain the discrepancy demands an effective population size the mitochondrial coding region is too small to support, regardless of era. A critic cannot borrow the plausibility of selection acting in a large, stable, modern population and transplant it onto a small ancestral one where the same mechanism has already been shown, quantitatively, not to reach far enough. The pedigree data close the remaining gap: if selection were distorting the mutation spectrum, the direct pedigree studies would be expected to show it, and instead Sigurdardottir and Howell both report the opposite. Background selection is real, well documented, and beside the point. If selection can't work then background selection definitely can't work. Background selection only works after purifying selection does and we have two studies showing us that the effective population size for selection to act upon the mitochondria was not enough. The low control-region diversity can not be explained by selection strength of today; and no amount of present-day selection can retroactively supply the population size the theory needed in the past. Both of these rescue devices are inadequate.

This brings us to time dependency, which basically just means that the mtDNA rate you calculate gets slower the farther back in time you measure it. Since evolution cannot find a slow clock in any species to match phylogenetic assumptions, they try to slow the current fast clock down by any means necessary, just so that it can touch the tip of the phylogenetic estimates would be good enough. One such attempt came from *Ho et al.* (2005): They analyzed several mitochondrial datasets, including primate D-loop/control-region sequences, and explicitly incorporated previously published human pedigree-derived rates. Time dependency became the tool of choice. *Ho et al.* (2005) did not merely acknowledge time dependency; they tried to mathematically model it. They assembled mitochondrial rate estimates ranging from very recent pedigree measurements to deep phylogenetic calibrations and proposed that the apparent rate falls with time according to a “vertically translated exponential decay curve.” For the primate D-loop/control region, that curve was then used to recalculate human and Neanderthal divergence times. The original *Ho* exponential curve however did not hold up as a general solution. In 2007, *Brent Emerson* specifically reanalyzed *Ho et al.*'s data. His paper's title leaves little ambiguity: ***“Alarm Bells for the Molecular Clock? No Support for Ho et al.'s Model of Time-Dependent Molecular Rate Estimates.”*** *Emerson* argued that the apparent long exponential decline was heavily affected by the particular calibration points, methodological assumptions, and datasets *Ho* had assembled. A subsequent mitochondrial study summarized *Emerson's* reanalysis particularly clearly: whereas *Ho et al.* had proposed that elevated rates persisted for roughly 1–2 million years, *Emerson's* reanalysis found “no evidence of elevated rates for events older than 100 kyr.” So essentially *Ho's* 2005 model and claim was that the observed fast rate becomes a gradual exponential decline which then reaches equilibrium at roughly 1–2 Ma. While just two years later it was falsified by *Emerson* 2007 reanalysis who showed that any detectable elevation disappears by 100 ka based on even the most favorable models. *Burridge, Crow, Fletcher, and Waters* in 2008 also agreed that *Emerson's* reanalysis found no elevated rates beyond approximately 100,000 years and questioned several of *Ho et al.* 's calibration points because some were themselves derived from molecular-clock assumptions. They independently undermined the long, multi-million-year tail required by *Ho's* original exponential curve. *Hans-Jürgen Bandelt* as well reviewed the controversy in 2008 and wrote that the *Ho* 2005 study was “beset with several problems” that had not been convincingly resolved. After reviewing the exchanges between *Ho* and *Emerson*, *Bandelt* asked what evidence remained for strong time dependence and answered: “Not much.” Much later in 2015 *Ho et al* tried to still defend the general phenomenon of time-dependent rate estimates, but they explicitly stated this time that the relationship was: “best described by a power law ... rather than by a translated exponential function as previously believed.” Notice the significance. The translated exponential function was the model proposed by *Ho et al.* in 2005. Ten years later, *Ho* and colleagues themselves finally admitted that this was not the appropriate mathematical description. They continued defending the existence of some time-dependent bias, but conceded that the original biological explanation could not account for its claimed magnitude. *Ho's* 2015 paper is especially interesting because it also backs away from another aspect of the original explanation. *Ho et al.* (2005) regarded purifying selection as the most convincing explanation for the declining apparent rate. But by 2015, He and his team explicitly acknowledged that studies had shown purifying selection was insufficient to explain the magnitude of the rate decline and made this quote in their published paper: “purifying selection alone is unlikely to provide a full explanation”. Let's look at a list of scientists who also all disagree with *Ho's* time dependent molecular clock idea. *Michael Woodhams* (2006) calculated that explaining *Ho's* curve entirely through deleterious mutations required implausibly large effective population sizes and undermined *Ho's* principal biological explanation of purifying selection. *Hans-Jürgen Bandelt* (2008) criticized the claimed exponential time dependence as a “tempest in a teacup.” Direct scientific commentary rejecting the importance and reliability of the proposed curve, although not a new empirical analysis. *Sankar Subramanian* and *David Lambert* (2011) found time dependence at only selectively constrained mitochondrial sites but “virtually” identical rates at synonymous neutral sites. They reject a general time-decay law for neutral changes and how much purifying selection can slow the D-loop. *Brent Emerson and Michael Hickerson* (2015) concluded that the hypothesized magnitude lacked sufficient theoretical and empirical support and that much apparent decline arose from artifacts and estimation biases. A broad, direct rejection of the strong TDMR hypothesis. *Emerson et al* (2015) used simulations and showed that BEAST could infer incorrect tree structures and underestimate effective population size. This demonstrated that the standard diagnostic test *Ho* used could fail to detect substantial bias.

8. The MRCA is not the first woman objection

The standard reply to any most recent common ancestor is that the MRCA need not be the first member of a species. Mitochondrial Eve had many female contemporaries; we accept this general point. Our claim is narrower and structural: the timescale on which coalescence to a single maternal lineage occurs is not free, it is set by the demographic history, and the two frameworks pay for it in very different currencies. For effectively neutral mtDNA the substitution rate is independent of population size, but the time to coalescence is not: it is a genealogical quantity that scales with the female effective population size, with the whole-sample maternal MRCA expected on the order of $2N_{ef}$ generations back (the pairwise expectation is N_{ef}). This is why the mainstream estimate places the mitochondrial MRCA deep, on the order of 200,000 – 300,000 years: reaching a single ancestral lineage from a standing population requires a very large number of generations for lineage extinction by drift to run to completion. The deep date is therefore not an independent observation but a direct consequence of assuming a large, long-lived ancestral population. Shorten the timescale without changing anything else and the coalescence does not complete.

There is only a single maternal MRCA who lived in a recent timescale according to the pedigree data. The single ancestral female must have passed through a severe recent bottleneck that collapsed all lineages into a single maternal ancestor. The standard model contains no such event: it posits a large, continuously distributed ancestral population and no recent global bottleneck, which is precisely why its coalescence runs deep. Our framework contains exactly such a bottleneck and creation date timeframe as a structural feature, not an auxiliary patch: the Flood reduces the maternal line to a handful of women a few thousand years ago, after which the post-bottleneck lineages diversify at the observed pedigree rate. The recent MRCA is not something our model must explain away; it is something our model requires. The evolutionary model requires the opposite and needs to explain based on the published evidence and dates obtained from it, how a MRCA can possibly land on such a date and their model cannot do so. Not a single simulation using assumed population size during that time can account for why this is the case because the world is supposedly already full of millions of women around the world and in isolated locations which would make a single MRCA to all humans alive today impossible. The fast pedigree rate compresses the post-coalescence diversification into a few thousand years, as Sections 5.1 to 5.4 show. The bottleneck supplies the coalescence event itself. The standard model can match the observed control-region diversity only by spending the deep-time budget on its own coalescent demands, and must then absorb the fast pedigree rate, the recent variant-age distribution of Section 9.1, and the recent growth onset of Fu et al. as separate facts requiring separate mechanisms. The recent-origin model obtains all of them from one event. We claim that given a recent timescale, evidence of a bottleneck is mandatory, and that our framework supplies one and theirs does not, and that on the criterion of explaining the most with the least, the bottleneck account is the more logical reading of the same evidence. We would also point to a broader pattern: across all animal taxa ever tested, though minimal, the germline pedigree-based mtDNA rates consistently exceed phylogenetic rates, including species classified as living fossils.

We read this as consistent with recent diversification. Mainstream researchers read the same pattern and invoke time-dependency, the proposal that the molecular clock slows down over time, and the authors who published those fast rates and shallow coalescence MRCA dates did not draw a recent biblical creation or flood bottleneck conclusion even though the numbers fall within those windows. This is not surprising; scholars trained only in evolutionary frameworks naturally read the data through that lens alone. We do not expect them to do otherwise, nor is their disagreement with their own conclusions or our interpretation of their results evidence against our model. What matters is that the published results are now published in the scientific record and available for independent interpretation. The data itself, the pedigree rates, the control-region diversity, and the decay kinetics of ancient DNA, are not in dispute. What differs is how they are read. The decisive test is explanatory power. Our model does not need to invoke anything, we let the evidence speak for itself, the data is correct and we point to it. The results land squarely on the biblical timeline as expected. The evolutionary model did not expect such a recent date for a MRCA and cannot force a bottleneck to account for the young dates, so they reconciled the discrepancy by invoking everything from time-dependent rates, saturation via parallel mutations, purifying selection, and demographic structure to try and explain away the paradox.

Our argument is not that these mechanisms do not occur or do not make an impact over vast time, but that they have no impact on our model and the evidence is clear on this. Mainstream evolutionary consensus needs these mechanisms to slow down the clock and should be modeled explicitly and tested against their own data, rather than used to attack our model. Their model has this problem, that is why they invoke such mechanisms in the first place, if this were not the case then clearly the paradox would have been resolved decades ago, yet it persists without any known resolution and every mechanism together does not close that gap and each new study only confirms and compounds the problem further. When the observed rate is taken at face value, the recent-origin account explains the mitochondrial signal and the demographic, historical, and biochemical record around it, with fewer independent assumptions than the alternative. By the criterion of parsimony, that is the stronger reading, and it resolves the apparent paradox rather than deferring it. By default this also means our model best explains the diversity of life on earth as well since diversity is explained through mutational differences which are based on mutation rates.

9. Convergent lines of evidence

In this section we set the mitochondrial estimate beside six further bodies of evidence that bear on the timing of human population history: the age distribution of human protein-coding variation, the arithmetic of population growth from a small founding number, the genealogical convergence of all living people, the abrupt and recent onset of the documented historical record, the internal chronology of the Biblical genealogies that match and flow into real world genealogies, and the measured decay kinetics of DNA itself. The mitochondrial clock remains the load-bearing, discriminating evidence, along with the decay rate of ancient DNA serving as a secondary clock. The additional lines are corroborating, not discriminating: independent lines of evidence sit comfortably in the same narrow window, and the recent-origin model offers the more parsimonious account of that convergence.

Statistical probability is the proper lens for weighing this. Each of these clocks listed below runs on different physics, mt-DNA substitution, decay kinetics, genealogical combinatorics, autosomal variant ages, demographic growth, and a textual chronology, and each could have returned almost any value across the roughly two to three hundred thousand plus year span the deep-time model allows. The recent-origin model stakes out a narrow target, a few thousand years wide, and predicts in advance that every clock should fall inside it. This makes the model highly susceptible to falsification.

The deep-time model makes no such prediction; it expects the mitochondrial and decay clocks to read in the hundreds of thousands of years and must, after the fact, explain each short reading away with a separate mechanism. While it cannot do even that, it also fails to obtain a close window to any of the dates it has claimed in the past. The dates have changed over tens of thousands to even over 100,000 years for Homo sapiens origins. Their view is unfalsifiable and their windows change frequently without any consequence. The fact that a half-dozen unrelated measurements all land in the same narrow few-thousand-year window is, on the recent-origin hypothesis, exactly what was predicted, and on the deep-time hypothesis just a rare coincidence that for them grows less plausible with every additional line of evidence added. From a statistical standpoint, the convergence of multiple independent kinds of evidence on a single narrow timeframe is precisely the signature of a true model, while the deep time framework requires an increasingly implausible series of coincidences to accommodate each new piece of data. The recent origin model is the more parsimonious account of the assembled evidence.

9.1 The recent origin of human protein-coding variation, and the onset of expansion

Fu et al. (2013) resequenced 15,336 genes in 6,515 individuals of European American and African American ancestry and dated 1,146,401 autosomal single-nucleotide variants. They estimated that roughly 73 percent of all protein-coding SNVs, and roughly 86 percent of those predicted to be deleterious, arose within the past 5,000 to 10,000 years. The deleterious class is the more telling of the two, because harmful variants are precisely the ones purifying selection removes fastest, so a large standing load of them is the signature of a population that expanded so recently that selection has not yet had time to clear it. The same study went further and dated the expansion itself.

From a demographic analysis of 1,351 European Americans and 1,088 African Americans, Fu et al. placed the maximum-likelihood onset of accelerated population growth recently and even states that using a demographic model incorporating that recent expansion, they estimate 73.2% of the protein-coding SNVs studied arose within the past 5,000 years. Another analysis of exome variation places the recent explosive-growth phase at roughly 205 generations ago, with extremely rapid growth in both the European American and African American modeled populations. *Tennessean* et al 2012 places the maximum-likelihood time for the onset of accelerated population growth was 5,115 years ago.

Both Fu et al. and Tennessean et al. reached similar conclusions using different analytical approaches from both exome and SNV data, which also coincide with pedigree-based analyses. Far beyond coincidence and again, evidence from alternative sources converging, this is consilience and one of the best ways we can validate our models' superiority. The *Tennessean* and *Fu* studies do not date the origin of humanity, and we do not claim that they do. The dates they are giving are the beginning of a rapid demographic expansion, and it lands within our biblical timeframe as expected. This is precisely the event our model predicts: a recent spreading-out of an already-founded population following the post-Flood bottleneck and the dispersal from Babel. Our model needs no such preceding interval: the expansion follows directly from the founding, and the 5,115-year onset falls around the post-bottleneck dispersal our chronology already expects. The pedigree analysis of this paper combines this data and uses it to answer two of the three biggest events in Biblical history, creation and the Tower of Babel dispersal.

9.2 Population growth from a small founding number

We begin with the founding number our model supplies after the Flood bottleneck: three couples, or six individuals. (*The Biblical account documents Noah and his wife but no additional children, so the eight total aboard the Ark reduces to three maternal lineages post-Flood*). To reach today's world population of roughly 8.2 billion in the 4,500 to 5,500 years our chronology allows requires an average annual growth rate of only about 0.39 to 0.48 percent. Rates in that range are unremarkable. They sit well below the global growth rates recorded through much of the twentieth century, which exceeded 1 percent per year for decades and peaked above 2 percent, and growth rates within this range have been observed in every variety of historical human populations, including tribal and frontier populations.

Growth certainly fluctuated, with famine, disease, and war pulling it down for short stretches, but the long-run average the model requires is modest by any historical standard. What is notable is the reverse calculation. If anatomically modern humans had been present for 200,000 years or more as evolution poses and had sustained even a very small positive average growth rate, the cumulative population, and the cumulative number of burials, would vastly exceed anything the demographic or skeletal record contains.

This tension is long recognized and is known as the hominin fossil gap paradox. The paradox rolls over into animal species as well. Mainstream demography holds that pre-modern growth was not modestly positive but very near net-zero for almost all of prehistory: high fertility was balanced by high mortality under probabilistic Malthusian constraints, with sustained growth beginning only at the Neolithic and accelerating agriculture on that account. We do not claim that growth arithmetic refutes deep time; the near-zero-growth model can absorb it regardless of evidence.

We include it because the recent-origin model reaches the observed modern population from a small recent founding number using ordinary, observed growth rates without any conditional interpretation involved. No extended epoch of stasis required thus no paradox, while the standard model must posit that stasis as a separate condition and that it occurred throughout most of history. The recent-origin account is again the more direct, and the growth onset it requires coincides with the 5,115-year expansion signal of Section 9.1. While evolution invokes an out of Africa scenario between 70,000 to 50,000 years ago, though earlier smaller groups left supposedly as early as 180,000 to 210,000 years ago.

9.3 The genealogical convergence of all living humans

Rohde, Olson, and Chang (2004) used mathematical computer simulations to model the genealogical ancestry of all living people under realistic assumptions about migration and population substructure. They found that the most recent common genealogical ancestor of everyone alive today, the most recent person from whom all of us descend through some ancestral line, likely lived just a few thousand years ago, on the order of 1500 BC or 3,525 years ago, and that the identical-ancestors point, the still earlier moment beyond which every then-living person who left descendants at all is an ancestor of every person now alive, fell at around 5,353 BC or 7,378 years ago in all of their models, including placing this MRCA outside of Africa. That window coincides with the mitochondrial and demographic estimates above. We present Rohde et al. as a third, independent source for two narrower purposes to illustrate that recent universal genealogical ancestry is mathematically possible. First, it shows that recent universal common ancestry of all living humans is not a peculiar creationist claim but a mathematical expectation, one which removes one common intuitive objection to a recent shared origin. Second, the recent-origin model is the one in which the genealogical and the genetic convergence coincide, rather than being separated by orders of magnitude like evolution; this makes our model far more parsimonious.

9.4 The sapient paradox and the onset of documented history

Anatomically modern humans are conventionally dated to roughly 200,000 – 300,000 years ago based on radiometric dating, yet the oldest known; medical text, written numbers, recorded mathematics, measurement systems, economic contracts, wheeled vehicles, evidence for complex civilizations, bureaucratic governments, territorial states, writing systems and papyrus writing material including recorded history itself all appear around the last 5,000 years or so, and continuous documentary history in all cultures all within roughly the last 5,000 as well. Colin Renfrew named this the sapient paradox: the puzzle of why behaviorally modern humans, if they existed for so long, left no record of the cumulative, documented culture they began producing in such density once they started. Mainstream answers with different scenarios such as climatic triggers, demographic thresholds, or even cultural-ratchet effects that supposedly held for tens of millennia and then released. We note, again carefully, that the term itself is framed from within a deep-time chronology; it is a paradox only on the assumption of the long preceding interval. That is exactly our point. On a recent-origin chronology there is no paradox to resolve, because there is no long silent interval to explain: documented history begins shortly after the founding population because the founding population is recent. The data, the abrupt and recent onset of the cumulative record, are agreed by everyone. What differs is whether one must additionally posit a cultural and cognitive ratchet that stayed locked for the preceding eighty-five percent of our species' supposed existence without direct historical evidence. The recent-origin model requires no such additional assumption, and here Occam's razor does real work. Where mainstream fails is prehistory, in our opinion they put radiometric dating before genetic dating. That is why all of these problems and paradoxes exist for their model and why they cannot get the genetic data to match the geologic data. Two different clocks telling two different stories will never align. For life, we test life. The age of the earth and rocks are a totally different topic that the secular community has bound their model together with. Since they are bound, they can never get a correct view of the past or coherent model without paradoxes.

9.5 Scriptural and traditional genealogies

The textual record is a further line. The biblical chronologies reconstructed from the genealogies in Genesis, place the creation of humanity and a Flood bottleneck just thousands of years ago. Compilations of the connected genealogical chains that descend from these accounts trace named lineages across this same span. For example; we have unbroken chains of genealogies in different places around the world, one such example is the lineage compilations maintained at the royal genealogical charts found on *The British Monarchy's Official Web Site* and on display at Hatfield House in Hertfordshire, England. These examples are just textual and traditional evidence and we weigh it accordingly. We include it because the framework this paper tests is the Biblical one, and the chronology internal to that framework places the relevant founding and bottleneck events squarely inside the window where the genetic and demographic lines independently converge. The agreement between an internal textual chronology and externally measured pedigree is the kind of correspondence a true chronology would be expected to produce and a false one would not.

9.6 Archaic hominins

Most attempts to interpret archaic age and divergence work from the entire mitochondrial genome, where the coding region can be affected by many different factors. We take the opposite approach and read the neutral compartment directly, using the maximum fixed differences in the same hypervariable segments used throughout this paper, clocked at the same pooled pedigree rate of one substitution per 19.947 generations, alongside an alternative generation time reflecting assumed and published archaic lifestyle. The table below presents the 16-year lower-bracket calculation, reflecting the earlier maturation proposed for these archaic populations, the shorter interval reflecting the earlier maturation and reproduction expected of these populations, so that the observed archaic diversity can be read against the biblical timeline. Majority consensus sequences were built from the complete Neanderthal mitochondrial genomes of Briggs et al. (2009), namely Feldhofer 1, Feldhofer 2, Sidrón 1253, Vindija 33.25, and Mezmaiskaya 1, who found Neanderthal mitochondrial diversity to be substantially lower than worldwide modern human diversity, and for the Denisovan panel consisted of FN673705, FR695060, KT780370 and KX663333, a deeply divergent archaic lineage. Distances were counted across the same aligned HVR1 and HVR2 window used above and measured against the Reconstructed Sapiens Reference Sequence, the reconstructed modern human matrilineal root. Because counts depend on alignment boundaries and the handling of indels and ambiguous bases, the working distances below should travel with their coordinates and processing rules.

On the archaic generation time

The shorter 16 year generation is not a free parameter chosen to fit the chronology; it follows from the developmental evidence for these populations. Synchrotron histology of Middle Paleolithic juveniles shows that most Neanderthal tooth crowns grew more rapidly than modern human teeth, producing significantly faster dental maturation and advanced molar eruption relative to recent human populations (Smith et al., 2010). Because molar eruption age across hominins and primates is a recognised correlate of the age at weaning and the age at first reproduction, faster dental development points to an earlier onset of puberty and reproduction, and therefore a shorter mean generation interval, than the 20 year figure used for modern humans. A 16 year generation is a conservative expression of that difference. It is offered here as a lower bracket.

The same pedigree clock can be turned on the archaic mitochondrial genomes. Under the recent-origin topology tested here, the Neanderthal and Denisovan control-region lineages are treated as branches descending from the reconstructed human root (the RSRS) and are clocked at the same pooled pedigree rate of one substitution every 19.947 generations. Because the archaic samples are ancient rather than living, we use a 16-year generation interval for these lineages.

A note on convention. The human pairwise distances of Sections 5.4 to 5.10 are tip-to-tip: the d differences separate two present-day lineages, each of which has accumulated d halved substitutions since their common ancestor, so the coalescence age is $(d / 2)$ times the rate times the generation interval. The archaic distances in this section are, within the topology tested here, root-to-tip: d is measured along the single archaic branch back to the human root, so the whole of d has accumulated on one lineage and the age is d times the rate times the generation interval. The two conventions are not interchangeable, and mixing them silently would make the archaic lineages look about twice as old as the human comparisons for the same measured distance. We therefore state both. Table 9 gives the root-to-tip ages used in the recent-origin test, and Table 10 restates the same distances under the symmetric tip-to-tip convention, so a reader who prefers to treat the archaic split exactly as the human splits were treated can read the comparison directly.

We read the neutral compartment directly, using the maximum fixed differences in the same hypervariable segments used throughout this paper, clocked at the pooled high-end pedigree mean of one substitution every 19.947 generations per lineage, with a 16-year generation interval reflecting the earlier dental maturation evidenced for these populations (Smith et al. 2010). Under the recent-origin topology tested here, the Neanderthal and Denisovan control-region lineages are treated as branches descending from the reconstructed human root.

Table 9 gives the root-to-tip ages; Table 10 restates the same distances under the symmetric tip-to-tip convention used for the human comparisons in Sections 5.3 to 5.8, so the archaic distances can be read under the same convention as the human comparisons.

Comparison	d	Root-to-tip age (years)
Neanderthal versus human root	22	7,021
Denisovan versus human root	25	7,979

Table 9. Archaic root-to-tip ages, d times 19.947 times 16 years.

Comparison	d	Symmetric tip-to-tip age (years)
Neanderthal versus human root	22	3,511
Denisovan versus human root	25	3,989

Table 10. The same archaic distances under the symmetric tip-to-tip convention, $(d / 2)$ times 19.947 times 16 years.

Under the root-to-tip convention, the Neanderthal lineage dates to approximately 7,021 years and the Denisovan lineage to approximately 7,979 years; under the symmetric tip-to-tip convention, the two fall to approximately 3,511 and 3,989 years, respectively. Either way, the archaic mitochondrial distances remain dramatically shorter than the hundreds of thousands of years assigned under conventional phylogenetic chronologies, which is the comparison of interest here. A fast-rate sensitivity calculation sharpens the point: using the Denisovan distance of 25 substitutions, the fastest single-pedigree control-region rate reported among the sources considered here, one substitution every 9 generations (Madrigal et al. 2012, pedigree 10), and a 16-year generation interval gives $25 \times 9 \times 16 = 3,600$ years root-to-tip. This is approximately 2.22 times younger than the 7,979-year estimate obtained from the pooled high-end rate, while still remaining far removed from a deep-time chronology. A pedigree-specific upper bound sharpens the point. Using the Denisovan distance of 25 substitutions, the fastest single-pedigree control-region rate reported in the sources considered here, one substitution every 9 generations (Madrigal et al. 2012, pedigree 10), and a 16-year generation interval gives 25 times 9 times 16, or 3,600 years root-to-tip, and 1,800 years under the symmetric convention. Compared with the 7,979-year pooled root-to-tip figure, the fast-rate estimate is about 2.22 times younger, which brackets the sensitivity of the archaic result to the choice of pedigree rate without ever approaching deep time.

For an example of this exact example in nature we take you to the water flea. *Daphnia magna*. It has an average mitochondrial base-substitution rate of about 8.7×10^{-7} per base per generation, among the highest directly measured in animals. Yet, it's within the genus species *Daphnia pulex* has a substantially lower rate despite the two species being related and having broadly similar morphology, physiology and life history. Mutation rates also differed by more than three orders of magnitude among individual *D. magna* genotypes. The precise molecular mutation causing the high *D. magna* rate remains unknown. The researchers suggested that differences in mitochondrial polymerases, replication, damage or repair must be involved. They also observed that *D. magna* has a smaller estimated effective population than *D. pulex*, which may permit weaker DNA-repair systems or higher-rate variants to persist; this is an evolutionary explanation rather than a demonstrated molecular cause. Note that this is also what we creationist state about Denisovan, Heidelbergensis and Neanderthal, that they also had smaller estimated effective populations, which permitted weaker DNA-repair systems or higher-rate variants to persist. So, ironically what they claim sped up the mutation rate in a current living species far beyond the normal rate is what we also claim about these small archaic populations of the past. For what caused this, just like *D. magna*, the precise molecular mutation causing the high mutation rate to begin in Denisovan and get inherited down the line to all branches of later subspecies remains unknown.

Similar to *D. magna*, several possible causes of the elevated mutation rate have been proposed; for further discussion, see *Budinsky (2026)*. The most probable candidates in the mtDNA we believe are variants shared only by Denisovan, Neanderthal and Heidelbergensis but not modern humans or chimpanzees. So we believe the strongest candidate is the mitochondrial variant ND4L N94S, caused by the nucleotide change m.10750A>G. This mutation changes amino acid 94 of the ND4L protein from asparagine to serine. All three

archaics have a derived S which is the amino acid serine. What makes it stand out is not simply that Neanderthals, Denisovans, and Heidelbergensis all carry the serine form, but that both chimpanzees and bonobos retain the same non-mutated ancestral asparagine also found in modern humans. That polarity strongly indicates that N94S arose only on the archaic human lineages rather than being an ancestral residue that modern humans later changed.

ND4L is also unusually relevant because it is part of mitochondrial Complex I, NADH dehydrogenase, the first major energy converting complex in the respiratory chain. Complex I controls electron flow into the respiratory system, contributes to the proton gradient used to manufacture ATP, and is also one of the principal sites associated with mitochondrial reactive oxygen species production. Therefore, even a modest alteration in this machinery could easily influence electron leakage, oxidative stress, mitochondrial turnover, or replication dynamics. That connection makes N94S particularly interesting to a mitochondrial clock hypothesis, because the “clock” is ultimately measured by the accumulation of mitochondrial sequence changes. If a lineage specific alteration in Complex I slightly changed the oxidative environment surrounding mtDNA or altered the rate at which mitochondria are damaged, replaced, and replicated, it could theoretically change the rate at which mutations become available for transmission and fixation. This evidence provides a biologically plausible mechanism that is strengthened further by previous computational work assigning m.10750A>G a MutPred score of 0.846, placing it among mitochondrial substitutions predicted to have relatively high functional impact.

The following calculation is presented as a high-rate sensitivity analysis, testing the effect of applying an observed high-end human pedigree rate to the archaic-hominin branch without invoking hypermutation. Human pedigree data include several high-end pedigree-specific mutation estimates extending across multigenerational genealogies. These estimates demonstrate that rapid accumulated rates can be observed across deeper pedigrees and can be applied to archaic *homo*.

These rates were 2.22 times faster than the pooled rate and by using them to explain Denisovan and Neanderthal diversity, it changes everything. Those ages of thousands of years drop dramatically without even invoking a mechanistic hypermutation cause. For example in *Madrigal* 2012, pedigree 10 results were one substitution every 9 generations (1/9) and, this was an observed rate in a pedigree 6 generations deep going back to a MRCA born 1846, 9 meioses across 3 typed subjects. We use this as an upper-rate sensitivity boundary since it demonstrates a sustained biological rate inherited through an entire pedigree, however for the Denisovan branch mutation rate we can only speculate on. This rate demonstrates mathematical compatibility under an observed high-end pedigree rate; So if after 6 generations the rate was still as observed as 1/9, this is a clear rapid germline recurring rate we can apply to a lineage that continues past 6 generations. This rate is deliberately selected from the highest end of the observed pedigree-specific estimates.

It provides us with a high-rate boundary test: it asks whether a rate actually observed in a multigenerational human pedigree is mathematically capable of accommodating the highest observed Denisovan control-region distance within the proposed recent chronology. Let's apply this observed rate to Denisovan who have the highest diversity differences at 25. Using the pedigree rate of 1/9 observed in *Madrigal* 2012 pedigree #10 to calculate how long these differences would take to accumulate after the bottleneck.

Using the Denisovan distance of 25 substitutions, a pedigree specific substitution rate of one substitution every 9 generations, and a 16-year generation time: $25 \times 9 \times 16 = \mathbf{3,600 \text{ years}}$. This is approximately 2.22 times younger than the 7,979 year estimate produced by the pooled 1 per 19.947 rate. Therefore, the Denisovan mitochondrial lineage coalesces with the reconstructed human root after approximately 3,600 years under this faster pedigree rate, making it approximately 2.22 times younger and much easier to explain in the YEC timeframe if we assume Denisovan were a new branch that formed after the flood.

The calculation demonstrates that an observed high-end human pedigree estimate can mathematically accommodate the Denisovan distance within the proposed recent chronology. Since we are confident that Neanderthal, Denisovan and Heidelbergensis all lived recently and on this same side of the bottleneck and can be corroborated by the genetic evidence, the population size evidence from Vaesen et al. 2019 regarding their

small population size and inability to survive for even 5,000 years, and the retrievable DNA from the *Sima de los Huesos femur*, including *Tennessen et al.* marker at 5,115 years for the assumed beginning of accelerated population growth and divergence and corroborated independently by *Fu et al.* looking at SNVs. Including the sheer lack of fossil evidence of their existence in their supposed long timeframe of existence.

The significance of this calculation is that it requires neither an invented mutation rate nor an unobserved biological mechanism. We can take what has been observed and apply it directly. Similar to pedigree 11 in Madrigal 2012 who observed a close one substitution every 10 generations, we have multiple examples in independent pedigrees of different lineages having much faster mutation rates. If this observed high rate existed in the early branch of Denisovan and was passed down to all subsequent generations and branches from them. Then explaining their diversity post flood becomes easy.

This calculation however is a sensitivity analysis rather than proof that Denisovans maintained this exact rate. The rate was measured directly in a multigenerational human pedigree and remained rapid across many successive maternal transmissions which even was a surprise to *Santos et al* (2005). It demonstrates the mathematical consequence of a rapid observed pedigree estimate in real time. When that empirically observed rate is applied to the deepest archaic mitochondrial distance, even the Denisovan lineage can be accommodated within the same recent historical framework as modern humanity. The archaic genomes therefore do not compel hundreds of thousands of years; they require only a single high mutation rate that already exists among tested lineages, precisely as pedigree studies and living organisms demonstrate today.

But this is not the only possible scenario either. Think of Eve's mitochondrial genome as the trunk of a tree. During the approximately 2,000 years between creation and the Flood, mutations accumulated in different maternal descendants. Eve did not need to possess all the diversity herself at the same time. Her daughters, granddaughters, and later female descendants formed separate maternal branches as diversity increased via mutations. By the time of the Flood, the human population would have contained many mitochondrial lineages, even though every one ultimately descended from Eve. The bottleneck then reduced that larger collection to the three maternal lineages carried by the sons' wives. Those three women would have differed from one another because their strictly maternal family lines had been separated for generations before the Flood. Whether Adam and Eve would be classified morphologically as *Homo erectus*, Denisovan, Heidelbergensis or anatomically modern *Homo sapiens* has no direct effect upon the mitochondrial calculation. Mitochondrial DNA is inherited maternally, so the relevant mitochondrial root is the founding maternal lineage. Under a single founding maternal-lineage model, the different archaic and sapiens mitochondrial branches would have arisen from that shared maternal root after creation.

Because people interbred before the biblical law was mandated, mitochondrial lineage and physical appearance would not necessarily travel together. A Neanderthal-looking individual could carry a maternal lineage relatively close to the lineage leading to modern human phenotypes. That is actually consistent with our reconstructed tree, which grouped the Neanderthal consensus more closely with RSRS and grouped Denisovan with Sima: ((RSRS,Neanderthal), (Denisovan,Sima)). This means "*archaic*" is not one mitochondrial family, it is a phenotype. Neanderthal and Denisovan diversity cannot all be placed in one special archaic lineage carried by just one maternal Ark woman. A better explanation is that variants later found in *archaic*-looking individuals may have been distributed across more than one of the three surviving maternal lineages.

Under this model, archaic-looking populations would survive for another 2,000 – 3,000 years after the Flood. Their mitochondrial branches would continue accumulating mutations during that period. Their later disappearance would not necessarily mean that their entire genetic contribution vanished. Two different things can happen:

- A maternal lineage can become extinct because the last woman carrying it has no daughters.
- The population's other nuclear DNA can survive through sons, mixed descendants, and interbreeding.

Thus, an archaic-associated mtDNA branch could disappear even while some ancestry from that population remained in later humans. Likewise, an archaic phenotype could disappear without the associated mitochondrial lineage necessarily disappearing at exactly the same time.

We have used both root to tip distance and pairwise distance calculations between lineages throughout this paper and we will do so again here. At a 20-year generation interval, the expected root-to-tip and pairwise differences after the 2,000 year pre-Flood period are: One substitution per 19.947 generations with expected changes on one branch at 5.01 with expected distance between two branches being 10.03. Thus, two women entering the Flood bottleneck could already have differed by approximately 10 substitutions at the pooled high-end rate, provided that their maternal branches began separating near the beginning of the proposed chronology. These are expectations rather than fixed outcomes; individual lineages could carry more or fewer substitutions because mutation is stochastic.

Under the present model, archaic-looking populations survived for approximately 2,000 – 3,000 years after the Flood when the ice age had ended. Their total elapsed history from creation to their final post-Flood period would therefore be approximately 4,000 – 5,000 years. If two maternal branches began separating near creation and both continued accumulating substitutions, their expected pairwise distances would be: 20.05 after 4,000 years total or 25.07 after 5,000 years. The pooled rate therefore provides a direct mathematical pathway to approximately 20 – 25 differences within the proposed 4,000 – 5,000 year interval. All diversity is captured within our timeframe.

So a distance of 20 differences at one substitution per 19.947 generations corresponds to approximately 3,989 years of divergence from the common maternal ancestor. A distance of 22 corresponds to approximately 4,388 years. Relative to a Flood occurring 2,000 years after creation, these values correspond to approximately 1,989 and 2,388 years after the Flood, respectively. Both fall within the proposed 2,000 – 3,000 year survival period of the archaic-looking populations. The two branch calculation tests whether the total observed separation between maternal lineages can arise within the proposed chronology. It does not establish that exactly half of the substitutions occurred on each branch. The ancestral reconstruction provides a complementary test by attempting to assign changes to particular parts of the mitochondrial tree.

Within the explicitly analyzed 658-bp HVR-I plus HVR-II interval, the reconstructed broader-*Homo* root differed definitely from RSRS at four positions, from the Neanderthal consensus at 17 positions, and from the Denisovan consensus at 11 positions, with additional ancestral positions remaining unresolved. The corresponding direct pairwise distances were 22 differences between RSRS and the Neanderthal consensus and 20 between RSRS and the Denisovan consensus.

These results show why root-to-tip and pairwise calculations should not be treated as interchangeable. The pairwise distance measures the complete path connecting two lineages, whereas the ancestral reconstruction indicates that the substitutions may be distributed unequally along that path. The symmetric calculation is therefore best understood as a chronological model projection rather than proof that exactly one-half of the observed differences occurred on each lineage. Nevertheless, the pairwise calculation remains appropriate for testing the total diversity-producing capacity of the model. If multiple maternal branches began diverging before the Flood, the mutations accumulated on both sides contribute to the distance ultimately observed between their descendants. The ancestral reconstruction then provides a second test of whether the inferred distribution of those mutations is plausible under the same chronology.

This model is best presented as variants later recovered from archaic-looking individuals who could have been distributed among more than one of the three maternal lineages that survived the bottleneck. One surviving woman could have carried a lineage related to the later Denisovan/Sima branch, while another could have carried a lineage related to the later RSRS/Neanderthal branch.

The sequences do not identify which historical woman carried which lineage, nor do they demonstrate that the sampled branches correspond exactly to the three women. They show only that such a distribution is compatible with the proposed branching model.

Supporting Data: Broader Homo Mitochondrial Root Reconstruction

Taken together, there are multiple lines of evidence that allow us to use the observed data to explain the diversity we see in both modern and archaic lineages and the genetic evidence does not place Neanderthals and Denisovans outside the history of humanity as remote evolutionary side branches far back into the distant past; instead, their patterns of ancestry, interbreeding, and shared genetic inheritance are consistent with genetically distinctive human populations existing within the broader recent historical framework of mankind.

9.7 Ancient DNA: reading the decay clock at a known temperature

The next line of independent evidence comes from the chemistry of DNA itself, and unlike the others it can be turned into a direct, falsifiable test and clock on a single specimen. DNA is not stable after death. It fragments through several measurable chemical pathways, dominated by hydrolytic depurination, whose rate follows the Arrhenius law and so is fixed by temperature (Lindahl 1993). For a specimen of known burial temperature, the surviving fragment length is therefore itself a clock, readable independent of the geological dating of the surrounding rock.

Allentoft et al. (2012) calibrated that clock directly, measuring mitochondrial DNA across 158 radiocarbon-dated moa bones and recovering an exponential decay with a half-life of about 521 years for a 242 bp fragment, a per-nucleotide rate of 5.50 times 10 to the minus 6 per year at 13.1 degrees Celsius, and an Arrhenius relation $\ln(k) = 41.2 \text{ minus } 15,267.6 \text{ divided by } T \text{ in kelvin}$. The mean fragment length after time t under first-order fragmentation is $L = 1 \text{ divided by } (k \text{ times } t)$, so the age implied by an observed length is $t = 1 \text{ divided by } (k \text{ times } L)$. We apply this to the specimen the deep-time framework treats as its showcase of extreme DNA survival, the Heidelbergensis Sima de los Huesos hominin femur (*NCBI still labels NC_023100.1*) from Atapuerca, Spain, conventionally assigned an age of about 430,000 years. Two facts make it an ideal test. See paper titled: *The clock that should not exist* (Nailor, M. 2026).

First, its burial temperature is known and was never frozen: the cave holds stable 10.6 degrees Celsius for most of the year, rising to about 13.4 degrees for roughly three months, and the excavators themselves describe it as the only non-permafrost site on Earth preserving Middle Pleistocene DNA. This is decisive for the present purpose, because it closes the cold-preservation escape that applies to the general case. Here there is no need to assume a temperature; the temperature is measured, known and well documented and it is consistently mild.

Second, the DNA is severely degraded, with recoverable fragments running from 30 to 45 bp and a mode near 30 bp, exactly the near-floor state in which the decay clock is most informative. We carry three sources of uncertainty honestly. The first is fitting uncertainty in the rate constant k itself. The second is the biological scatter implied by the low fit of the Allentoft model, whose R-squared of 0.39 means that about 61 percent of preservation variance comes from soil chemistry, pH, hydration, and other taphonomic factors rather than age and temperature.

The third is that the two recoverable fragment lengths are the two ends of a bracket: the longest fragments, near 45 bp, are a ceiling, since the true mean length is shorter and so yields the youngest age the data allow, while the 30 bp, sit at the detection floor of the method, meaning they are older. We therefore report each scenario as an interval between these two ends together with its midpoint, treating the midpoint as a convenience summary of the bracket rather than a precise point. Because the cave's thermal regime is known, we restrict attention to the two rate scenarios that fit it. The temperature-weighted rate, $k = 3.93 \text{ times } 10 \text{ to the minus } 6 \text{ per nucleotide per year}$, derived below from the cave's own seasonal cycle, is the proper central rate and supersedes a flat constant 10.6 degree; a faster rate is also warranted because the location in the cave is perpetually damp and wet, which is poorer for preservation and accelerates decay even faster. We set aside the flat-average and the ideal-preservation scenarios, since neither matches a wet, warmer temperature-cycling cave.

Applying $t = 1$ divided by $(k \text{ times } L)$ at the 45 bp ceiling and the 30 bp floor gives the intervals and midpoints in Table 8.

Scenario	Rate k (per nt per yr)	45 bp (ceiling)	30 bp (floor)	Midpoint
Combined, seasonal + poorer preservation (central)	6.23×10^{-6}	3,570 yr	5,350 yr	4,460 yr
Fast (poorer preservation, +1 sigma, flat 10.6°C)	5.28×10^{-6}	4,200 yr	6,300 yr	5,261 yr
Temperature-weighted (seasonal, mean preservation)	3.93×10^{-6}	5,655 yr	8,483 yr	7,069 yr
Central (Allentoft mean, flat 10.6°C)	3.33×10^{-6}	6,700 yr	10,050 yr	8,342 yr
Slow (best preservation, -1 sigma, flat 10.6°C)	2.10×10^{-6}	10,600 yr	15,900 yr	13,228 yr

Table 8. *Kinetic age readings for the Sima de los Huesos femur at the cave's measured temperature, for the three preservation scenarios that fit a wet, temperature-cycling cave: a fast poorer-preservation rate, a temperature-weighted seasonal rate, and the two combined.. Each scenario is given as an interval from the 45 bp ceiling (younger bound) to the 30 bp detection floor (older bound), with the midpoint reported as a summary of that bracket.*

The temperature-weighted rate is obtained from the cave's seasonal cycle: nine months near 10.6 degrees ($k = 3.35 \text{ times } 10 \text{ to the minus } 6$) and three months near 13.4 degrees ($k = 5.67 \times 10^{-6}$) give a time-weighted effective rate of 3.93 times 10 to the minus 6 per nucleotide per year, about 17 percent faster than the flat average. Read at the cave's own temperature, the femur dates to a midpoint of about 5,261 years under the faster, poorer-preservation rate that a wet, saturated cave warrants, and to about 7,069 years under a conservative temperature-weighted only rate, *with the full ceiling-to-floor brackets shown in Table 8.*

We can also combine the two into one scenario: the seasonal temperature cycle and poorer-than-average preservation (+1 σ). At that rate: 45 bp equals 3,570 years at 45 bp, 5,350 years at 30 bp, **midpoint 4,460 years**. We take this scenario's true rate reading as it takes not only the cave's natural conditions into account but also takes the cave's temperature into account as well.

Both results are just a few thousand years, and both leave the claimed 430,000 year age an extreme outlier, more than sixty times. Run in reverse, survival of 45 bp fragments for 430,000 years would require a rate constant about 65x slower than the known rate of decay based on a constant 10.6 degrees, which the Arrhenius relation reaches only at a sustained burial temperature near minus 9.8 degrees Celsius.

The cave has never been a freezer, thus not a preservation time-machine. We are explicit that the kinetics fix a young window of roughly 3.5 to 7 thousand years rather than a single year, and that our preference for the earlier, post-Flood reading follows from the cave's wet conditions rather than from the rate constants alone. This is, in any case, the prediction our creation model made in advance: hominins such as *Homo heidelbergensis* lived recently, on this side of a global bottleneck, and their molecular remains should read young when measured by any clock that does not borrow its calibration from the deep-time framework.

The DNA decay clock is exactly such a clock, and it reads young. The decay calculation is presented as a biochemical stress test on the assigned age derived from radiometric dating. What would falsify this data is a measured, strontium to strontium compared to carbon dating directly of the fossil compared to one another for congruency and then cross referenced with the collage data and see which one corroborates it. With the temperature objection closed by the cave's own thermometer, this line of evidence is an independent clock we can use as even further parasimonious evidence, and it points where the rest do.

9.8 Ancient population size and the paradox

Vaesen et al 2019 is another study that also independently confirms this data. Taken directly from 'The Neanderthal Extinction Dilemma' paper by Thomas, (2019) *"Four researchers from the Netherlands recently published the results of their computer-modeled human populations in the journal PLOS ONE. The findings show that small Neanderthal population sizes would have caused them to become extinct in just 10,000 years. How did Neanderthals survive the 400,000 years they were supposedly on Earth?"* This is a logical question and once we add *Homo erectus* who supposedly lived for 2 million years and *Homo heidelbergensis* who existed for approximately 500,000+ years, the problem is compounded and made far worse for the mainstream view. In

Smithsonian Magazine titled; Rethinking Neanderthals, we read *"Paleoanthropologists generally agree that **Neanderthals lived in groups of 10 to 15, counting children**. That assessment is based on a few lines of evidence, including the limited remains at burial sites and the modest size of rock shelters. Also, Neanderthals were top predators, and some top predators, such as lions and wolves, live in small groups."* This is further corroborated in Anthropology News March 2021: *"Modern archaeological research has picked away at one of the trickier problems in understanding Neanderthals: How many of them lived together? High-resolution sites (where sediments accumulated slowly and short occupations can be discerned) confirm that groups likely contained **no more than 20 individuals**".* This was confirmed the following year by Skov et al 2022.

In regards to the Vaesen et al data, the data shows they enter extinction risk at a population size of 50 by 10,000 years, but what about realistic smaller population sizes? Since we know Vaesen's 2019 equations we can apply it to these tribal population numbers that archaic man existed in. When applied, you will quickly notice that the numbers favor the Biblical model and greatly hinder the evolutionary model even further. The additional stochastic extrapolation of the demographic framework used by Vaesen et al. (2019) was performed using strictly isolated bands of fifteen individuals, a typical tribal size including children, with each band held at that ceiling. Using an independently implemented Vaesen-derived individual-based simulation, five thousand replicate bands were followed for up to 5,000 years while retaining the same baseline demographic schedule and the same population-dependent Allee relationship used in the preceding simulation. Under a moderate Allee condition ($A = 10$), every band became extinct: the median extinction time was approximately 134 years, the central 80 percent of extinctions fell between roughly 81 and 239 years, and essentially no band persisted beyond 500 years. Strengthening the Allee effect to $A = 15$ again produced extinction in all replicates, with a median extinction time of approximately 113 years. Put simply, a band of fifteen does not behave like a band of fifty with proportionally less time on the clock: when numbers remain this small, demographic chance itself becomes the dominant hazard.

A handful of deaths, an unfavorable sex ratio, too few reproducing females, or several unsuccessful reproductive years can remove a large fraction of the entire breeding pool at once, while inbreeding and reduced reproductive opportunity compound the decline. Within this extrapolated framework, a strictly isolated band of fifteen therefore has essentially no prospect of persisting even one thousand years, let alone several thousand. These values extend Vaesen's published demographic results into a still smaller population range. The reason Denisovans and Neanderthals persisted longer than these few-hundred-year limits is that random female exogamy operated between a network of tribes that periodically encountered one another (Skov et al., 2022; Slon et al., 2018). Isolated small bands are fragile, therefore archaic groups needed the exogamy network the genetics shows, to survive any extended periods of time at all. The genetic evidence provides a mechanism by which communities this small could persist substantially longer than completely isolated bands. Skov et al. (2022) found that the best-fitting models for the Chagyrskaya Neanderthals involved communities of approximately twenty individuals in which an estimated 60 to 100 percent of reproductive females were migrants from other communities. This is considerably more extensive gene flow than an assumption of only one unrelated female entering every fourteen years, and it therefore replaces that unsupported parameter in the present model.

Direct evidence that genetically differentiated archaic groups also reproduced when they encountered one another comes from Denisova 11, the first-generation offspring of a Neanderthal mother and a Denisovan father (Slon et al., 2018), which demonstrates Neanderthal to Denisovan interbreeding. This factor boosted the survival of lineages in which inbreeding alone would have driven extinction in fewer than twenty generations. In this independently implemented extension of the demographic framework of Vaesen et al. (2019), the surviving communities were represented as effective bands of about twenty individuals, since a strictly fifteen-person band cannot approach the multi-millennial range even under recurrent gene flow, while unrelated reproductive-age females were permitted to enter at random from neighboring groups. Scientists estimate that at any given time the total population of Neanderthals across Europe and Asia was quite small, generally numbering between 5,000 and 10,000 individuals alive simultaneously (Degioanni et al., 2019). Female reproduction was allowed to begin at age sixteen, consistent with the demographic assumptions used in the present analysis.

Twenty-six thousand replicate bands were simulated (*imagine 26,000 parallel universes, each with its own Denisovan band*), with persistence evaluated through 4,000 years. An exchange frequency averaging approximately one unrelated reproductive-age female every 10.5 years substantially prolonged the survival of these otherwise highly vulnerable communities, increasing median persistence roughly four to five times relative to fully isolated bands, while still resulting in effectively universal eventual extinction. The median extinction time was approximately 597 years, with the central 80 percent of extinctions occurring between roughly 224 and 1,531 years. Approximately 25.5 percent of bands persisted beyond 1,000 years, 4.6 percent beyond 2,000 years, 1.9 percent beyond 2,500 years, 0.8 percent beyond 3,000 years, and only about 0.32 percent beyond 3,600 years; the single longest-surviving band among the twenty-six thousand replicates reached roughly 3,975 years.

Recurrent female exogamy can therefore greatly extend the possible persistence of an extremely small community by introducing unrelated reproductive-age females and relieving unfavorable local demographic and inbreeding conditions, yet under these assumptions it does not remove the fundamental instability of maintaining a community at only about twenty individuals. Persistence approaching 3,600 years lies deep within the extreme survival tail of this simulation and remains vanishingly rare rather than representing a normal demographic outcome; it is not an absolute mathematical cutoff, and a small number of simulated bands even persisted somewhat beyond 3,600 years, but these were very rare.

At the 3,600-year mark, here's what the simulation actually leaves standing: At year 3,600: In roughly 25,920 of those universes, the band is dead (extinct). In roughly 75 – 80 of those universes, the band is still alive. Those maximum 80 surviving bands hold about 840 people total across all 80 universes combined or ~0.3% (averaging ~11 people per band). Next we have *Kelly Harris and Rasmus Nielsen* in 2016 in the paper titled: *The Genetic Cost of Neanderthal Introgression*, which revealed that rather than Neanderthal ancestry declining gradually at a constant rate over thousands of generations.

Harris and Nielsen's additive-effects model predicted that most of the reduction would have occurred rapidly during the first 10 - 20 generations after interbreeding. In their simulation, an initial Neanderthal ancestry proportion of approximately 10% fell below 3% within 20 generations, a period of roughly 260 - 580 years using their assumed generation length of 26 - 29 years. After this initial decline, the removal of Neanderthal DNA became much less efficient, and the remaining ancestry declined far more slowly throughout all subsequent generations. All this independent evidence helps us explain the paradoxes in evolution such as the sapient paradox, population paradox, and the hominid fossil gap paradox which refers to the lack of well-preserved hominid fossils from a million-year period in the fossil record.

All of these paradoxes are not paradoxes at all in our model, they do not even exist as a problem because all of this evidence fits perfectly with what we would expect to find. These predictions are another line of evidence and one that Dr. Jeanson from AiG is expounding upon with his new Y chromosome data that aligns well with mtDNA mutation rates and known historical events (Jeanson 2022).

9.9 Pedigree testing regions

To determine when a MRCA lived using a pedigree substitution rate, we have to look specifically at the regions tested. Most substitution rate studies that look for a MRCA date in humans and different species use the D-loop or the hypervariable regions inside it, not the entire mtDNA or coding region. Those regions add factors that cause additional complications that are easily avoidable by focusing on the better effectively neutral D-loop region. Plus, a mutation rate is not population wide, it is lineage specific so it tells you very little in comparison. Geneticists know this, and below is direct evidence of that looking at various mtDNA studies over the decades. Connell et al. (2022) explicitly observed that pedigree research traditionally concentrated on the control region or small coding-region segments because of the expense and labor involved in producing complete mitogenomes by Sanger sequencing. The historical literature therefore provides a long and comparatively continuous record of directly observed control-region variation. The studies listed below document this repeated regional focus.

PEDIGREE DATA SETS

Hauswirth & Laipis 1982
Olivo et al. 1983
Wilson et al. 1997
Gocke et al. 1998
Baasner et al. 1998
Hühne et al. 1998
Laipis et al. 1988
Ashley et al. 1989
C M Koehler et al 1991
Howell et al 1996
Bendall et al 1996
Mumm et al 1997
Soodyall et al 1997
Parsons et al 1997
Jazin et al 1998
Kavar et al. 1999
Lutz et al. 2000
Cavelier et al 2000
Sigurdardottir et al 2000
Bowling et al. 2000
Savolainen et al., 2000
Heyer et al 2001
A T Bowling et al 2002
Cristina Luis et al 2002
Forster et al. 2002 †
Hill et al. 2002 †
Kavar et al. 2002
Sekiguchi et al. 2003
Howell et al 2003
Brandstätter et al. 2004
Pfeiffer et al. 2004
Bini & Pappalardo 2005
Santos et al. 2005 †
Szibor et al. 2007
Rose et al. 2007
Kim et al. 2007
Asari et al. 2008
Fridman & Gonzalez 2008
Moreau et al. 2009
Forster et al. 2010
Klüttsch et al 2010
Moreau et al. 2011
Madrigal et al 2012
Kurushima et al. 2012
He et al. 2013
Giuliani et al. 2014
Hudson et al. 2017
Shedko 2017
Andy C Dell et al 2020
Connell et al. 2022
Menéndez et al 2023
Árnadóttir & Helgason 2024

REGION ANALYZED

D-loop (control-region)
D-loop (control-region)
HVR1, including heteroplasmy at nucleotide 16355
Control region and cytochrome-c-oxidase intergenic regions
Control region, including maternal inheritance in CEPH families
HVR1 + HVR2
D-loop
D-loop (control-region)
D-Loop (Control region)
D-Loop (Control region)
HVR1
HVR1
D-Loop (Control region)
HVR1 + HVR2
HVR1 + HVR2
D-loop, two variable terminal regions of 378 bp and 310 bp
HVR2
HVR1 + HVR2
HVR1 + HVR2
HVR1
D-Loop (Control region)
HVR1 + HVR2
D-Loop (Control region)
D-Loop (Control region)
HVR1 + HVR2 of the control region
D-loop, HVR1 381-bp hypervariable segment
D-loop (Lipizzan maternal lines)
HVR1, including nucleotide 16291 heteroplasmy
D-Loop (Control region) Pooled studies
D-Loop (Control region)
D-Loop (Control region)
HVR1
D-Loop (Control region) + HVR1
HVR3
D-Loop (Control-region) fragment spanning nucleotides 16531–261
HVR1 + HVR2
HVR2
HVR1 + HVR2 in mother–child pairs
HVR1 + HVR2
D-Loop (Control region)
HVR1
HVR1 + HVR2
HVR1
HVR1 & HVR2
Full mtDNA + control region
D-Loop (Control region), including HVR1 and part of HVR2
D-Loop (Control region)
D-Loop (Control region)
D-Loop (Control region)
Entire mtDNA, with separate estimates for HVR1
D-Loop (Control region)
Entire mtDNA, Coding, Control (D-loop), HVR1

10. Cherry Picking

Critics of Biblical creation are not fond of pedigree studies, they say that they inflate the rate and we need to only account for unobservable long-term phylogenetic assumption rates only. Thus they say we are the ones cherry picking by not only using them, but specifically single generation pedigree studies as that. We can understand this criticism, but it is not true. In this study alone we evaluated pedigree depth explicitly and separately examined the subset based on consensus-level sequence differences while removing all single and even double generation pedigrees from both pools.

The main reason we use pedigree studies is because they follow the exact principle of what good science is all about; testable, repeatable observable evidence without any assumptions. The results speak for themselves and have for decades and have been shown to line up perfectly with known historical bottlenecks. We do not need to invoke any alterations to explain the diversity we see based on the timeline in our model. We believe that the only reason anyone would criticize this observable pedigree method is only if it produces results that go against what they personally believe to be true (evolution), which in this case it does and the authors of these studies themselves are a great example of that. They cannot fathom why the results do not reach evolutionary timelines and have since their discovery been an irreconcilable paradox where only speculation can close the gap.

The fact is, the entire mtDNA undergoes purifying selection because 90% of it is coding. Geneticists need to factor that in including population size. With the D-loop, neither of the variables matter and do not need to be factored in. That is why the region is selected for these studies and why it's superior. The control region is non-coding and non-transcribed into protein, so its sites are under weak purifying selection making it what geneticists call "an effective neutral region". So since substitutions there are closer to neutral, it means the count you observe is closer to the true mutational input, not a number that selection has already pruned or not pruned. The coding region is the opposite: synonymous sites aside, most nonsynonymous changes are removed by purifying selection before you ever see them, so the coding region's *observed* substitution rate is a selection-suppressed underestimate of the mutation rate. This is not fringe; it's the standard reason molecular geneticists treat coding and non-coding sites separately and why the D-loop's hypervariable segments are called hypervariable and why they are isolated for substitution rate studies when looking for a MRCA. Trying to combine the two rates and regions is a categorical error. Shared inheritance is not a shared mutation rate. Linkage governs how a molecule is *transmitted*, it says nothing about how fast different sites *mutate* or how selection filters them afterward. The D-loop is the right *window* for reading the substitution rate, even though it's inherited as part of one linked molecule. The only thing that would cross over is background selection affecting the D-loop and we have already covered that and why it is not a problem at all in our model in section 7.

Identification of the remains of King Richard III by King et al. (2014) is another study critics sometimes point to as evidence against a fast mitochondrial substitution rate. But that interpretation is not only logically weak; it misreads the study itself. King et al. explicitly adopted the high pedigree-based rate reported by Parsons et al. (1997), approximately one control-region substitution every 33 maternal transmissions, for their own statistical analysis. They wrote: "*To obtain the likelihood under H1, we require the mtDNA mutation rate, and in this case high estimates are conservative.*" They then stated: "*Because this suggests a higher rate than other published estimates, and is based on genealogical data, we used it to derive a probability of 0.52 for no mutation in 19 meioses.*" In other words, using the Parsons rate alone as they did, the probability of observing zero control-region substitutions across Richard III's there was a 52% probability. Meaning there was over a 50% chance of not seeing a mutation across just the 19 meioses. So why should the absence of a D-loop substitution over just 19 transmissions be considered surprising evidence against a fast pedigree rate? It should not be. Furthermore, King et al. only observed a single whole-mitochondrial-genome difference, but that substitution occurred at position 8994 in the coding region, not in the D-loop. Thus, it is not a D-loop rate study and critics who cite this study as evidence against a fast D-loop substitution rate are attempting to make the absence of an event statistically meaningful when the authors themselves calculated that such an absence was the most probable outcome, even under the very fast pedigree rate they adopted. Far from refuting a fast control-region rate, the Richard III results are entirely compatible with one.

The reality of the data tells us something fascinating. When we compare humans to chimpanzees regarding genetic differences, we see something that should not exist. Despite substantially greater standing mitochondrial diversity within chimpanzees than in humans, with comparative studies reporting chimpanzee mtDNA nucleotide diversity more than threefold higher than that observed in humans (Wise et al., 1997; Yu et al., 2003), and evidence from great ape pedigree analyses indicating that the present day chimpanzee germline mutation rate per year may be approximately 1.5 times faster than the corresponding human rate (Besenbacher et al., 2019). How can chimps have higher diversity within the species, and a higher mutation rate, yet have less differences accumulated in the chimpanzee line than the hominin line? That's right, the polarization of the human, chimpanzee mtDNA COI differences reveals an asymmetry in accumulated substitutions. The confidently polarized sites are distributed in a ratio that also matches this paradox in mtDNA. Across all 67 polarized mtDNA COI differences, approximately 67 differences between humans and pan are distributed by 30 substitutions to the chimpanzee branch and approximately 37 to the human branch. The same pattern is found in a four-taxon outgroup polarization of the mitochondrial genome outside the control region, using complete human, chimpanzee, gorilla, and orangutan RefSeq sequences, 809 human–chimpanzee differences could be polarized by agreement between both outgroups. Of these, 411 were assigned to the Homo branch and 398 to the Pan branch (Andrews et al., 1999; Horai et al., 1995; Xu & Arnason, 1996). Both results are the opposite expectation by extrapolating present day pedigree rates across the entire period of divergence. This anomaly was further noticed and expounded upon by Besenbacher et al. (2019) who estimated that the present day chimpanzee yearly germline mutation rate is 1.42 ± 0.22 , and human mutation rate as 1.50 ± 0.10 , yet noted that genome wide DNA comparisons make the chimpanzee branch only about 2 percent longer than the human branch. Confronted with this discrepancy, the authors stated explicitly that *“a slowdown in mutation rate in the human lineage must be recent”* and concluded that the *“human rate has recently decreased significantly”* (Besenbacher et al., 2019, pp. 288–289).

Moorjani et al. (2016) independently found remarkably similar accumulated substitution totals for the chimpanzee and human branches, 0.621 percent and 0.633 percent, respectively, **with the human branch actually longer**. This makes no sense, since according to them; *“life histories may have been fairly similar for much of their evolutionary history”* (Moorjani et al., 2016, p. 10610). Thus, the approximately 30 chimpanzee versus 37 human COI allocation is not merely inconsistent with a constantly faster chimpanzee clock; it parallels a recognized genome wide problem for constant rate extrapolation. The conventional resolution explicitly requires the present day human mutation rate to be historically unrepresentative, with the human slowdown arising relatively recently rather than characterizing the entire human lineage. This begs the question; if the human mutation rate has **slowed down recently**, yet it is too fast today to align with evolutionary phylogenetic dates. What chance do they possibly stand being able to reconcile these problems by invoking an even faster rate in the past? This compounds their problem even further.

The deeper problem for evolution is not merely that one mutation rate has been revised, but that the molecular clock itself randomly changes according to the shifting timescale, lineage, and calibration method used to read it. In mtDNA specifically, Henn et al. (2009) documented the typical discrepancy of nearly an order of magnitude between directly observed pedigree rates and rates inferred from human, chimpanzee phylogenies, even noting that the phylogenetic rate had often been preferred *“without theoretical justification for the phylogeny based rather than the pedigree based rate”* (p. 217). Ho et al. (2005) likewise found that primate mitochondrial data **required** the rate to shift over time and concluded unequivocally, *“Our results show that it is invalid to extrapolate molecular rates of change across different evolutionary timescales”* (p. 1561). Once that concession is made, defenders of conventional chronology are cornered and cannot have it both ways. They cannot invoke an unobserved historical time dependency slowdown, purifying selection, bottlenecks, recurrent mutation saturation and calibration dependent corrections whenever directly measured rates produce chronologically unacceptable results, yet dismiss accelerated or historically variable rates as illegitimate the moment creationists examine the same evidence. Those mechanisms may be legitimate considerations to help them resolve one issue, but the burden of proof must be symmetrical: every proposed correction is an admission of a failure of expected evolutionary results and a failure to actually demonstrate a quantitatively sufficient answer, not selected from a lost list of probabilities then invoked after the fact all because the observed clock gives them the wrong answer.

Moreover, when an assumed fossil or archaeological chronology is used to calibrate a slower genetic rate, the resulting molecular date cannot honestly be presented as wholly independent genetic confirmation of that same chronology. The observed substitution rates do not match what we would expect if humans had always been mutating at their present-day rate. To explain the difference, the conventional model must therefore assume that human mutation rates were much faster in the past and only changed in the recent past, even though those earlier rates were never directly observed and the rates today already run too fast for them. Our model is simple, we invoke that rates we observe are the true rates and that some people groups have both faster and slower rates. Evolution cannot make such a claim, they require a much slower rate to match phylogenetic rates, yet at the same time a much faster rate in the past to explain the discrepancy in diversity. Two contradictory things at the same time.

A reasonable question is, why the present analysis concentrates on the mitochondrial control region, particularly the standardized 610 bp HVR-I + HVR-II window, instead of using mutation rates calculated across the entire mitochondrial genome. The answer is not that whole-mitogenome data contradict or give us unfavorable results compared to the control-region results. On the contrary, applying the whole-mitogenome pedigree rates to comparable studies produces the same qualitative pattern. For example, using *Árnadóttir* et al. (2024) rate and the average worldwide diversity yields a recent linear projection, whereas the rare maximum pairwise distance yields a substantially older value. Take their whole mtDNA rate which works out to approximately: 4.25×10^{-6} mutations per bp per generation. Converting the whole-genome rate. Across 16,535 bp: $4.25 \times 10^{-6} \times 16,535 = 0.0703$ - So that is approximately: 1 whole-mtDNA mutation every 14.2 generations per maternal lineage. At a 20 years/generation time: $14.23 \times 20 \approx 285$ years. What does that give us in regards to when mitochondrial Eve lived based on the average diversity? Well, assuming *Árnadóttir*'s rate is an average, then the average diversity should fall around the Biblical timeframe. Let's test that and see if it is true. The 7,098-genome dataset (Kim & Schuster, 2013) published an average pairwise difference of 39.8 bases. Applying the exact same rate and using the published confidence limits on the mutation rate gives approximately 5,546 to 5,786 years. The same dataset contained a maximum pairwise difference of 123 bases. The corresponding rate-only sensitivity range is approximately 17,140 to 17,882 years using the average rate. Just like using their HVRs average rate of 1/42.6 you end up with 15,336 years. This is literally the same pattern, and the same solution. You use average rates and you reach the average diversity we see in the world today, right in the Biblical timeline. To reach the highest rare max diversity, you use the highest rare maximum rates. Nothing changes. So we do not ignore the data, nor cherry pick around it, it's obvious we chose the region that is less affected by mechanisms like population size and purifying selection since they make better clocks and the geneticists agree and do the same.

The purpose of restricting the primary analysis to a predefined HVR window is therefore comparability and reduced protein-coding and unknown population size constraints. Under the largest human pedigree study yet conducted, the whole-mitogenome central rate places the mean worldwide pairwise diversity at approximately 5,664 years and the rarest maximum diversity at approximately 17,503 years using that central average rate. The independently standardized HVRs calculation of the same study places the maximum at approximately 15,336 years. Therefore, the recent projections reported here are not created merely by ignoring the coding genome or entire mtDNA. They emerge in both the selected HVR window, D-Loop and an entire-mitogenome cross-check at a similar same ratio.

The central empirical question is consequently not whether the whole mitochondrial genome has been ignored. It has not. We selectively choose the D-Loop for the same reason geneticists have and because they have a long record of direct pedigree-based investigation. Control-region pedigree studies extend back for decades and provide a comparatively large, historically continuous body of directly comparable maternal-transmission data. While whole-mitogenome pedigree studies do provide important complementary evidence, they are unfortunately much more recent research and are also not interchangeable with a rate estimated for a defined HVR-I/HVR-II or D-Loop window. A whole-genome estimate combines sites with substantially different mutation behavior, such as the coding regions. This choice is consequently based on regional matching and comparability of the pedigree evidence, and limited studies. That imbalance in the available evidence is a methodological reality, not an evasion. The control region provides the deepest pedigree history, the largest body of directly comparable observations, and a window not affected by protein-coding constraint or population size.

The whole mitogenome nonetheless supplies a much more powerful independent test of whether that choice manufactured the result. It did not: when the analysis is expanded from 610 bases to 16,535, the same essential relationship remains, the mean worldwide diversity projects to only several thousand years, while the rare maximum lies approximately three times farther back. This demonstrates why the statistical center of global diversity must not be confused with its most exceptional tail end. A valid analysis must account for both, but it need not pretend that a rare maximum represents the typical pairwise separation within humanity. Across two distinct mitochondrial compartments, using independently calculated rates from the same extensive pedigree study, the central pattern survives the change in sequence window. The conclusion is therefore unavoidable: the recent signal is not an artifact created by cherry picking and selecting the D-loop or HVR's, it remains visible when the entire mitochondrial genome is allowed to speak for itself and we can answer both the average and maximum diversity using observed pedigree results in both the control region and entire mtDNA pedigree studies. For this paper we chose the better of the two clocks and that is far from cherry picking.

10.1 Whole-mitogenome high-end pedigree results

For readers who wish to see whether the same general pattern appears when the analysis is extended beyond the D-loop or HVRs to the whole-mitogenome rate, we will do the same thing we did in this paper and examine the upper-end of the directly observed pedigree results. In this case the max diversity is 123 max differences in the entire mtDNA. With far fewer whole-mitogenome pedigree studies available to pull from than control-region studies and studies that do not always measure precisely the same type of event. It is therefore necessary to distinguish the raw appearance of a new heteroplasmic variant from a mutation that reaches homoplasmy and becomes an accumulated consensus-level substitution. With that distinction clearly recognized, most of the fastest pedigree rates are heteroplasmic. These usually run fast, so we will use Ding et al. (2015) who Dr Jeanson used to obtain a mutation rate from. We will do the same, but go one step further. Note first that *Ding et al.* did not actually publish a rate based on 63 de novo mutations after 333 transmission events. *Dr. Jeanson* (2015) derived that number from Ding's aggregate Table 1. *Ding et al.* analyzed mitochondrial sequences obtained from lymphocytes of 2,077 Sardinian participants, including 333 parent-child trios. Their analysis distinguished homoplasmic sites, at which only one allele was detected in the sampled mtDNA, from heteroplasmic sites containing more than one detectable allele. Heteroplasmies were required to have a minor allele frequency of at least 4%. Among the 333 children, *Ding et al.* reported 7,273 homoplasmic variant instances relative to the revised Cambridge Reference Sequence, of which 7,238 were also found in the corresponding mothers. This *Ding*-only analysis gives a rate per genome of 0.1892 or 1 mutation every 5.29 generations. This rate was actually very high and resulted in a MRCA of 6,150 years.

Here is how the math works; using 123 differences divided in half you get 61.5. Then multiply times 5 generations you get 307.5 generations. With a 20 year generation time you land directly on 6,150 years. However Jeanson pooled two other pedigree studies with *Ding et al.* (2015). Rebolledo-Jaramillo et al. (2014) and Guo et al. (2013). Both only supplied transmission events and zero observed mutations. This slowed Jeanson's rate down to the point he needed to use a 15 year generation time to land exactly on 6,000 year Eve.

However, *Ding et al.* was unsure if all 100% of these homoplasmic mutations were truly germline or if some were somatic, the rate itself may not be 100% accurate if some are somatic. Jeanson directly states this in his paper in 2015: 375–378. *"To confirm germline transmission in the future, the DNA sequences from at least three successive generations must be sequenced to demonstrate that variants were not artifacts of mutation accumulation in non-gonadal cells."* Since his pedigree was only a single generation, there is a chance the rate is higher than it truly is. This is not much of a problem however, since human mitochondrial pedigree studies demonstrate that variants can be reproducibly detected and persist across maternal generations, and multiple studies show us what rate survives and what does not. We will look into these studies and apply the results directly to *Ding et al.* 2015 total and then go even one step further and apply that to a future untested assumed deeper pedigree of multiple generations where the rate continues to slow down.

Rebolledo-Jaramillo et al. (2014) confirmed 44/44 higher-frequency heteroplasmy calls (100%) and 16/18 lower-frequency calls after independent reamplification; **88.89%**.

Zaidi et al. (2019) confirmed 133/150 higher-frequency calls and five selected putative de novo variants. 133/150 = **88.67%**. Sanger validation of high-frequency heteroplasmy calls. The remaining 17 occurred in or near low-complexity regions, which were excluded.

Likewise, the persistence fractions calculated from a more recent study, Liu et al. (2022) following an ancestral cohort of heteroplasmies, 29 of 33 persisted through the next transmission. A **87.88%** retention, followed by 89.66% the third generation before stability. This means dyad pedigrees lose 11.52% in the first generation, while triad pedigrees lose another 10.34% in the following before stabilization.

We take the total observed first generation retention percentages of 88.89%, 88.67% and 87.88% = **88.48%**

Now that we see a very consistent survival percentage, let's apply this to *Ding* et al, raw rate and remove some variants that were counted as homoplasmic. With an **88.48%** retention, we retain 55.74 event-equivalents practically, about 56 mutations if using whole events. So we can claim that 7 of the 63 are hypothetically somatic.

Applied to 123 pairwise differences, the constant-rate, two-branch calculation would yield approximately: 367.16 generations. Now remember, Jeanson used a 15 year generation time. This lands on 5,507 years ago, the date of Noah's flood in the *Septuagint*. We however will not go below an 18 year generation time for the general population. Doing so lands on mitochondria Eve living 6,609 years ago. Essentially the same as *Parsons* results.

But let's not stop here, remember what Jeanson stated earlier? *"To confirm germline transmission in the future, the DNA sequences from at least three successive generations must be sequenced to demonstrate that variants were not artifacts of mutation accumulation in non-gonadal cells."* With *Liu et al. (2022)* data, we can conduct a sensitivity analysis by applying a further 89.66% retention stage, equivalent to a 10.34% loss, before the observed variant frequencies stabilize, as some variants drift below the detection threshold while others persist across successive maternal transmissions as shown by *Árnadóttir et al. 2024* three unite bottleneck data.

With an 88.48% first-stage retention, 55.74 event-equivalents remain from the original 63. Assuming a further 89.66% retention through the next transmission stage, approximately 49.98 event-equivalents remain. This produces an effective rate of 0.15009 retained events per whole mitogenome per maternal transmission, or approximately one event every 6.66 generations per lineage. Applied to 123 pairwise differences in a two-branch constant-rate calculation, this yields approximately 409.77 generations. At an 18-year generation time, the resulting linear mitochondrial Eve projection lived approximately 7,376 years ago. Perfectly within the biblical timeline, even after multiple generations of mutational loss through drift or possible misclassification of somatic variants as inherited events.

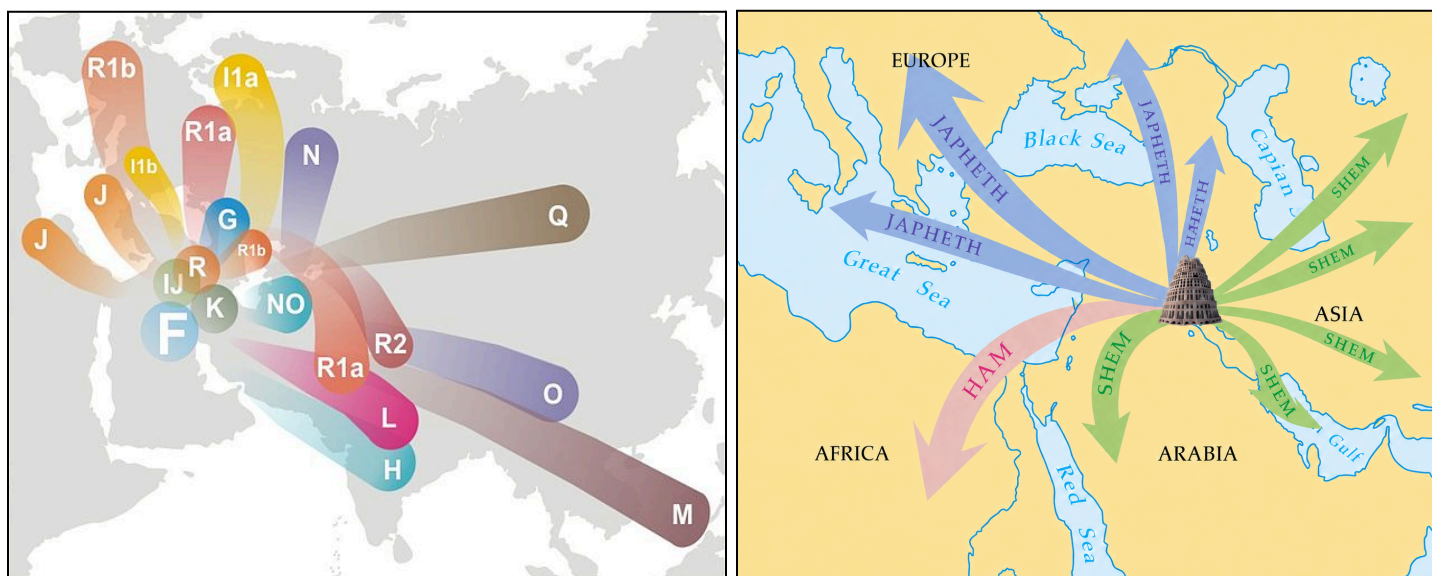
This sensitivity analysis we have simulated should not be attributed to Jeanson or presented as a revision of his published calculation. Jeanson retained all 63 aggregate homoplasmic discordances inferred from *Ding* et al. 's Table 1 and treated them all as true germline events, yielding a *Ding*-only point estimate of 0.1892 mutations per mitogenome per maternal transmission. The present analysis takes a different and deliberately more conservative approach. It does not assert that every excluded event was necessarily somatic; rather, it asks whether the result remains within the proposed recent-timeframe model after allowing for loss of apparent events or loss of their eventual contribution to persistent lineage differences through a stated sensitivity assumption. Because *Ding* et al. did not sequence three successive maternal generations for these particular discordances, their ultimate germline persistence cannot be established directly from that dataset alone. Our adjusted calculations are therefore our own conditional projections, not *Jeanson's* conclusions and not a replacement for the original *Ding* et al. observations. They show that the inference does not depend solely on retaining the maximum possible count of 63 events and that entire mtDNA and D-Loop results concur with one another.

11. Reading the convergence: Consilience

Figure 10 later shows consilience: the convergence of independent kinds of evidence on a single conclusion. The four mitochondrial pedigree estimators are the lines that most cleanly discriminate, and together they place the maternal coalescence between roughly 3,000 years for the mean pairwise distance and roughly 7,600 to 7,800 years for the deepest pair. The ancient-DNA reading of the Sima de los Huesos femur is a second, specimen-level test that reads young where the standard chronology assigns 430,000 years. The remaining lines do not by themselves discriminate, but each is accounted for by the recent-origin model directly and by the standard model only with an added mechanism: saturation and time-dependency for the pedigree rate, the farming revolution known as the Neolithic expansion to account for the sudden flood of rare gene variants and the moment population growth switched on, hunger and disease holding numbers flat to make the population math come out right, a slow buildup of culture over thousands of years to explain why nobody wrote anything down for so long, and, for DNA that supposedly lasted hundreds of thousands of years in a cave that never froze but sat at about 10.6 degrees Celsius, a fragmentation rate far slower than any yet measured. The recent-origin model explains the whole set with one founding-plus-bottleneck event; the standard model explains it with a series of separately posited conditions. Reading the chart from the top down, each marker is a central estimate and each bar the range reported or modeled around it, plotted in years before present.

The mitochondrial analyses provide complementary estimates based partly on shared rate and diversity inputs; they should not be treated as statistically independent measurements. Their central values span approximately 2,892 to 7,589 years in the principal pooled-rate calculations, with additional region-specific and fixation estimates reported separately. Figure 10 displays these estimates alongside demographic calculations, historical observations, molecular-decay scenarios, and scriptural chronology anchors. Not all plotted entries are independent or equivalent forms of evidence.

Below them sits the Tennesen et al 2012 marker and Fu et al at 5,115 years, the maximum-likelihood onset of accelerated population growth, with *Tennesen* et al corroborating the bar spanning sometime between 5,000 to 10,000 year window in which that study placed the bulk of rare protein-coding variants. These are mainstream results produced inside the standard chronology. What makes its timeline worth noting is that it falls where a bibbically recent founding event would put it, and that the standard model reaches the same place only by adding the Neolithic expansion as a separate explanation. Two independent studies, two independent results landing on the same recent timelines where creationist expected it to land and far from evolutionary expectations. The rise of haplogroups forming and diverging from this middle eastern area is yet another confirmation bringing the evidence together to form the bigger picture that coincides with exactly how the bible describes.



Figures 8 & 9: Figure 8 on the left shows the haplogroup formation location and migration pattern (M. Zlojutro, 2008). Figure 9 on the right shows the biblical location and story of Babel dispersion.

The population-growth bar is different in kind again. It is not a measurement but an arithmetic requirement: the span needed for three reproducing couples to reach 8.2 billion at sustainable rates of increase. Its lower end necessarily meets the Flood-bottleneck anchor at the foot of the chart, because the two are the same quantity approached from opposite ends.

The same caution applies to the two square markers at the bottom, the Flood bottleneck and the Creation window. This is not evidence. They are the model's predictions, placed on the chart so the reader can see for himself whether the independent estimators land on them or somewhere else entirely. The ancient-DNA evidence occupies its own block and needs to be read carefully. The dashed moa bound, marked with a diamond, is not a date for any human specimen. It is the calibration standing behind everything to its right: the radiocarbon-dated interval across which the 521-year half-life was actually measured. It appears on the chart because a decay constant is only as trustworthy as the range over which it was observed, and a reader is entitled to see how far the Sima estimates reach beyond that range rather than being asked to take the extrapolation on faith. The two Sima de los Huesos readings are shaded as a single specimen-level test rather than as two independent results, because they are the same femur read under two different assumptions: a fast or poorer-preservation reading at roughly 5,261 years, and a temperature-weighted reading at roughly 7,069 years. Neither is remotely compatible with the 430,000 years the standard chronology assigns to the specimen, and the discrepancy is not a matter of degree but a factor of sixty or more. Candor requires noting what the two readings do to the burial framing, however.

Vaesen et al. (2019) independently confirmed the key demographic point: small archaic populations are expected to disappear, not persist for tens of thousands of years let alone hundreds of thousands of years. The evidence therefore supports the biblical creationist view that aligns with these new Sima de los Huesos findings of recent collagen from a supposedly 430,000 year old heidelbergensis hybrid, that archaic humans were recent, isolated branches of mankind, not ancient evolutionary side branches enduring through deep time. The fast-preservation reading at ~5,261 years sits comfortably in the post-Flood window; the temperature weighted reading at ~7,069 years does not, since it would place the femur before the Flood and raise the further question of how such a deposit survived in situ.

We take this scenario's midpoint as our central reading for the reasons given in Section 9.6, and we flag the tension rather than let the shading obscure it. The shaded band at roughly 5,000 to 7,600 years is the convergence window itself: the interval on which the discriminating estimators independently settle. Its force lies not in any single marker, several of which are individually weak, but in the fact that lines of evidence with no methodological connection to one another, a pedigree mutation rate, a decay constant measured on extinct birds, and a population calculation, have no obvious reason to agree and yet do. Consilience of this kind is the ordinary currency of historical science, and on the criterion of explaining the most with the least, the recent-origin reading is the most parsimonious. That is the claim Figure 10 below is meant to support, and it is all it is meant to support.

The case for pedigree-derived mutation rates and particularly for direct measurement in the mitochondrial D-loop/control region, does not rest on a single study, laboratory, or methodological assumption. Across decades of mitochondrial research, investigators have repeatedly described pedigree methods as direct, assumption-light measurements of mutation, while simultaneously recognizing the control region as exceptionally informative for resolving recent genealogical and population history.

These acknowledgments are especially significant because they occur throughout a literature in which the much faster pedigree rates frequently conflict with conventional phylogenetic timescales in which the authors all adhere to. The methodological strengths of the approach therefore do not arise from advocates of a recent chronology imposing a preferred clock upon the data; they are stated repeatedly within the mainstream literature itself.

Authors have further argued that population size does not determine the neutral evolutionary rate, that purifying selection cannot readily explain away the control-region signal, and that the pedigree - phylogenetic discrepancy is genuine rather than merely an artifact of selection. Consider the statements from the literature itself:

- **Greenberg, Newbold & Sugino 1983; the paper that first localized the hypervariable segments and showed the noncoding region outruns the coding region.** *"The level of sequence divergence significantly exceeds the reported estimates of divergence in coding regions. Two particularly hypervariable regions have also been defined."* (abstract, *Gene* 21:33 to 49).
- **Santos et al. 2005;** We read, regarding the D-Loop control region: *"based on minimal assumptions"* and *"the failure of most phylogenetic approaches to consider the effects of population dynamics, admixture, and migration"*.
- **Howell and Bogolin Smejkal et al. 2000; control-region hypermutation.** *"The human mitochondrial genome evolves rapidly, particularly so within the 1.1-kb noncoding control region or D-loop."*
- **Heyer et al. 2001;** *"The mitochondrial genome, and its **control region (CR) in particular**, is the most analyzed DNA sequence in **human evolutionary studies**."*
- **Meyer, Weiss & von Haeseler 1999; control region framed as the central marker.** *"This study provides a comprehensive survey of the complex pattern of nucleotide substitution in the **control region** of human mtDNA, which is of **central importance to the studies of human evolution**."* (*Genetics* 152:1103 to 1110).
- **Excoffier & Yang 1999;** *"Mitochondrial D-loop hypervariable region I (HVI) sequences are widely used in human molecular evolutionary studies, and therefore accurate assessment of rate heterogeneity among sites is essential."* (*Mol. Biol. Evol.* 16:1357 to 1368).
- **Salas et al. 2000;** *"Variation in human mitochondrial DNA (mtDNA) has been used to infer the origin and migration patterns in human populations. mtDNA analysis has been focused mainly on the first hypervariable region (HVI)."* (*Eur. J. Hum. Genet.* 8:964 to 974).
- **Elson, Turnbull & Howell 2004;** *"The mitochondrial genome has been the most widely used system for the investigation of the evolutionary history of our species. **It has been the system of choice because of its high rate of sequence divergence and its uniparental, maternal inheritance**."* (*Am. J. Hum. Genet.* 74:229 to 238).
- **Soares et al. 2009; coding region carries a selection signal the control region lacks.** *"We here confirm a modest effect of purifying selection on the mtDNA coding region"* and described their comparison as being supported by *"**a neutral clock based on synonymous mutations alone***. Their detailed analysis concluded that the almost-linear relationship between synonymous substitutions and control-region changes indicates that the control region cannot be strongly affected by purifying selection.
- **Gupta et al. 2023; approximately 300,000 human genomes.** *"Heteroplasmic indel rates are orders of magnitude lower next to poly-C stretches in coding regions, suggesting negative selection in these regions."*
- **Howell et al. 2007;** *"For most sites in the control region, the relative rate of substitution was similar to the rate of neutral evolution"*.
- **Gunnarsdóttir et al. 2011; complete human mitogenomes.** *"We observed a significant excess of variable positions in the control region ... which can be attributed to weaker functional constraints."*

- **Payne et al. 2013; direct HV2 versus MT-CO3 comparison.** Control-region low-level variants were approximately **3.3 times** more frequent than variants in the similarly sized coding-region amplicon. The authors explain that “these substitutions may be tolerated during transmission, unlike coding region variants that undergo strong negative selection during transmission.”
- **Soares et al. 2009; purifying-selection comparison.** “The virtually linear relation between the accumulation of synonymous mutations and control-region mutations suggests that the control region cannot be greatly affected by purifying selection”.
- **Sanchez-Contreras et al. 2023; largest high-accuracy multi-tissue catalogue in its study.** “Clones ... distributed relatively uniformly across the mtDNA coding region, but with a striking clustering of variants in the mCR (mtDNA Control region).”
- **Torres-Gonzalez et al. 2026; direct indel comparison among mitochondrial regions.** “The D-loop appears to be the preferential site for indel mutagenesis.” In human data, indels were enriched **2.9-fold in the D-loop** but depleted to **0.6 of expectation in protein-coding sequence**, which the authors interpreted as higher coding constraint and high human D-loop mutability
- **Williams et al. 2013; independent human-brain sequencing study.** “Single nucleotide variants accumulate fastest in the control region.”
- **Howell and Bogolin Smejkal et al. 2000; control-region hypermutation.** “The human mitochondrial genome evolves rapidly, particularly so within the 1.1-kb noncoding control region or D-loop.”
- **Howell et al. 2003** on the pedigree/phylogenetic gap being real and not a selection artifact of the region: their line that “there is a real discrepancy” between the two rates, and their statement that “selection has not preferentially distorted the pedigree mutation spectrum relative to the phylogenetic one.” ... “selection isn't what's driving the control-region signal.”
- **Tamura/Nei-era methodological literature on why the CR is used.** “because of its extremely rapid evolution, which provides opportunities for resolution of genealogical history”. The full context specifically says that its rapid evolution permits genealogical resolution and divergence estimation even among populations.
- **Wang & Boles 2006; explicit statement of why HVRs are well suited.** “Due to maternal inheritance, lack of recombination and a high polymorphic density, the mtDNA control region hypervariable regions are well suited”.
- **Sigurðardóttir et al. (2000);** “The mutation rate of the mitochondrial control region has been widely used to calibrate human population history.” “It is clear that pedigree studies provide a direct estimate of the mutation rate μ .”
- **Parsons et al. 1997:** “The rate and pattern of sequence substitutions in the mitochondrial DNA (mtDNA) control region (CR) is of central importance to studies of human evolution and to forensic identity testing.”
- **Warren 2000:** Warren frames the whole enterprise around the D-loop precisely because it is non-coding and fast-evolving, and reports his rate “across the two hypervariable regions.” “the control region is the region of choice for direct-count rate studies,” in the author's own design.
- **Madrigal et al. 2012; framing of the noncoding region and the two-rate split.** “Estimates of mutation rates for the noncoding hypervariable Region I (HVR-I) of mitochondrial DNA (mtDNA) vary widely, depending on whether they are inferred from phylogenies (assuming that molecular evolution is clock-like) or directly from pedigrees.” (Am. J. Phys. Anthropol. 148:327 to 333)

- **Kivisild et al. 2006;** "protein-coding genes are under negative (purifying) selection, since their mutation spectra are characterized by a pronounced predominance of synonymous substitutions over non-synonymous ones ($Ka/Ks < 1$)."
- **Ramos et al. 2013; weaker functional constraint in the control region.** Because it does not encode proteins or RNAs, "less stringent functional constraints act on this portion of the mitochondrial genome."
- **Liu et al. 2022; four-generation human pedigrees.** The authors describe "the control region, which may be subject to less stringent selection pressure."
- **Howell, Kubacka & Mackey 1996; empirical pedigree evidence.** "The evolution of the human mitochondrial noncoding D-loop is both more rapid and more complex than is revealed by standard phylogenetic approaches."
- **Henn et al. 2009; why pedigrees can avoid phylogenetic-rate distortion.** "Phylogenetic analyses are expected to be affected by homoplasy and back mutation, especially at hot spots, but pedigree studies are not." They also found that genealogical rate estimates converge on the pedigree rate, not phylogenetic
- **Soares et al. 2013; pedigree rates and ancestral-polymorphism bias.** "The higher estimated mutation rates are obtained in pedigree studies where ancestral polymorphisms are not an issue."
- **Jazin et al. 1996; coding-region heteroplasmy filtered by selection.** "By contrast, heteroplasmy in coding regions was not detected. The lower level of heteroplasmy in the coding regions is indicative of selection against deleterious mutations."
- **Wei et al. 2019; region-specific selection in human pedigrees.** "We analyzed different functional regions of the genome and found evidence indicating region-specific selection for or against heteroplasmic variants." Their results demonstrate that observable mtDNA variation depends on genomic function, making coding-region counts less direct reflections of mutation input.
- **Stewart et al. 2008; direct experimental evidence for coding-sequence filtering.** "We report a rapid and strong elimination of nonsynonymous changes in protein-coding genes; the hallmark of purifying selection." This is experimental mammalian germline evidence, showing the same pattern. The coding region is under purifying selection while the control region is not, thus making the control region superior.
- **Heyer et al. 2001; second line, distinct from the one you have.** "confirming earlier findings of much greater mutation rates in families than those based on phylogenetic comparisons."
- **Connell et al. 2022; whole-mitogenome pedigree comparison.** "As expected, the mutation rate for the coding region was found to be smaller than that reported for the control region."
- **Madrigal et al. 2012; direct HVR-I pedigree estimates.** HVR-I rate estimates "vary widely, depending on whether they are inferred from phylogenies ... or directly from pedigrees."
- **Sigurdardottir et al. (2000), it is, rather, a phylogenetic rate estimate that may be biased."**

The pattern is remarkably consistent. Pedigree studies directly observe mutations as they arise between known maternal generations, avoiding the deep-time calibration points required by phylogenetic clocks. The control region, meanwhile, is repeatedly selected precisely because of its rapid evolution, high polymorphic density, maternal inheritance, lack of recombination, and capacity to resolve recent genealogical relationships. **Ingman et al. 2000 is the citation aimed at your thesis.** They deliberately moved away from the control region toward the coding region for phylogenetics, precisely because control-region homoplasy and hotspots rates do not align with evolutionary timelines.

Convergence of independent age estimators on a recent demographic window

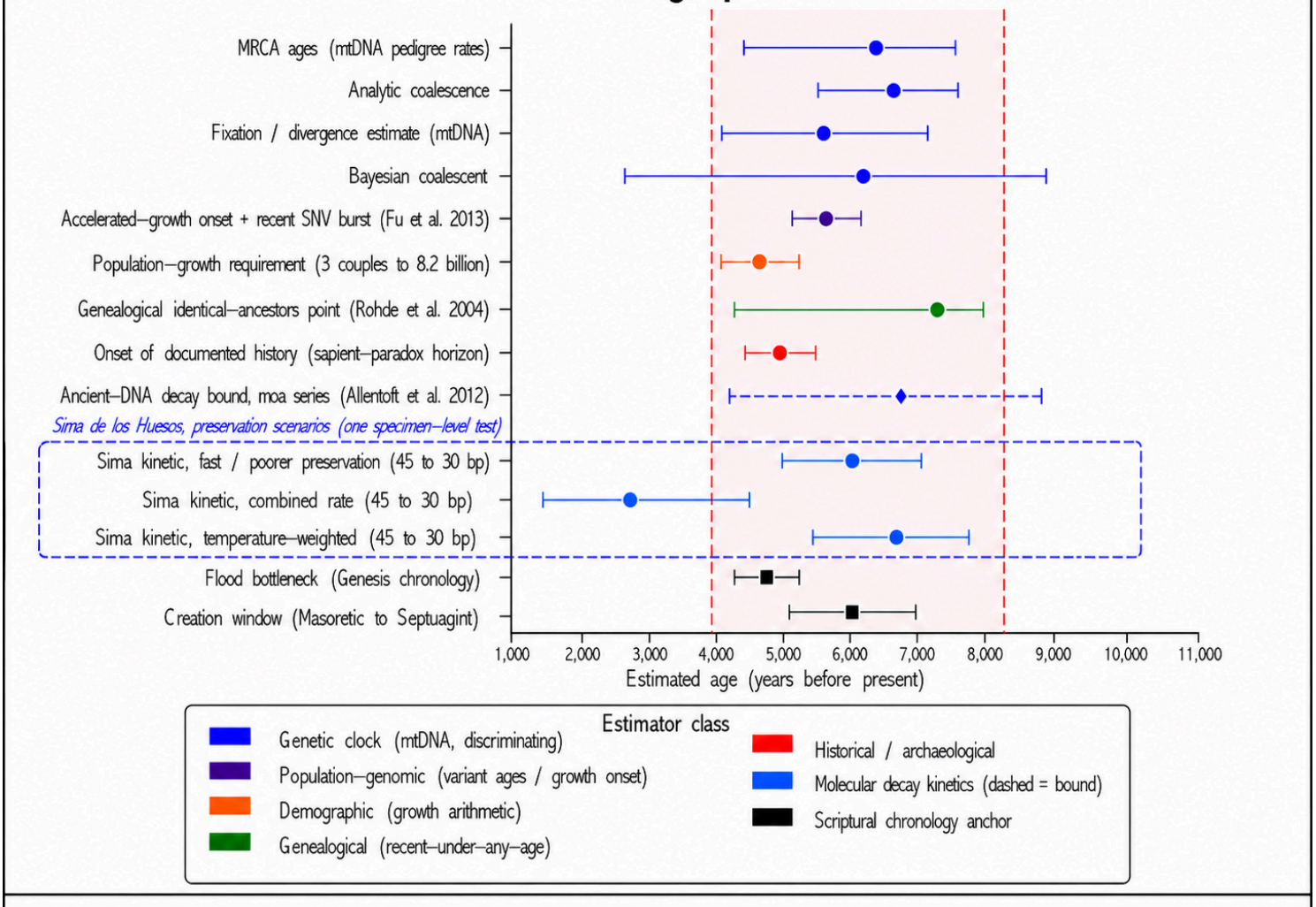


Figure 10. Independent age estimators plotted against a recent demographic window. Each marker is a central estimate; each bar is a range, plotted in years before present. Bar types are not equivalent and are distinguished as follows: solid bars are reported confidence or credible intervals from the cited studies; open bars are modeled or arithmetic spans derived in this paper (see Sections 5, 6 and 7). Discriminating estimators. The mitochondrial estimators (Sections 5.1 to 5.4) are the genetic clocks that discriminate between the competing chronologies. Non-discriminating estimators. The Fu et al. marker is the 5,115-year maximum-likelihood onset of accelerated growth, with the bar showing the reported 5,000 to 10,000 year variant-age range; this is a mainstream result obtained within the standard chronology and is plotted for position, not as independent support. The population-growth bar is an arithmetic requirement rather than a measurement: the post-bottleneck expansion span needed to reach 8.2 billion from three couples. Its lower end coincides with the Flood-bottleneck anchor by construction, since the two express the same quantity from opposite ends, and it therefore adds no independent confirmation. Ancient-DNA block. The dashed moa decay bound (diamond marker) is not a specimen date but the calibration behind the kinetic readings: the radiocarbon-dated range across which the 521-year half-life was measured. The two Sima de los Huesos readings are shaded as a single specimen-level test, not as two independent results, being the same femur evaluated under two assumptions: a fast or poorer-preservation reading (~5,261 years) and a temperature-weighted reading (~7,069 years). Both are incompatible with the 430,000 years assigned to the specimen by the standard chronology, a discrepancy of roughly sixty-fold. The fast-preservation reading falls within the post-Flood window; the temperature-weighted reading falls before the Flood anchor and is discussed in the text. Model predictions. The two scriptural anchors, the Flood bottleneck and the Creation window, are shown with square markers at the bottom. These are predictions of the recent-origin model, not evidence, and are plotted so the reader can judge whether the independent estimators fall on them. Shaded band. Approximately 5,000 to 7,500 years, the interval on which the discriminating estimators independently settle.

12. Beyond the Pooled Mean: Toward a Global Human Mitochondrial Rate

This study covers a lot, but it leaves out our personal views regarding what the average global mean most likely is. Based on the data; (Budowle et al 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 8.3).**” So to obtain the average differences for the African samples of the database, we will test this using the two most popular creationist starting dates and 14.1 as the African American average to see how the data accommodates it.

Populations	# Matches/ # Comparisons	Freq ($\times 10^{-3}$)	Avg. # Different Nucleotides $\pm$ SD
<i>Within group</i>			
Pairwise Comparisons			
African American	24/11026	2.18	14.1 $\pm$ 5.8
African (Sierra Leone)	61/6105	9.99	13.1 $\pm$ 5.6
US Caucasian	669/182106	3.67	7.9 $\pm$ 3.7

Figure 11. From Budowle et al 1999 Table 3.

Starting at the oldest date of creation in the Septuagint (7,600 years ago), and the same 20 year generation time the required rate lands on one substitution every 53 generations, which is almost identical to Santos et al., 2005 filtered rate (1/53.5), yet even this slow rate accommodates the African average diversity we see.

For the 6,000 year creation timeline with the same 20 year generation time, the rate would be one substitution every 42.6 generations, almost perfectly on Árnadóttir et al. 2024, results of (1/42). The relevant observation is that the rate demanded by the chronology is not invented or biologically unprecedented; it lies directly within the range already measured in living human pedigrees.

Remarkably, the substitution rate of 1/42.6 required to reproduce the high African American mean in 6,000-year chronology closely matches the approximately 1/42 HVR estimate derived from Árnadóttir et al. (2024), the largest pedigree-scale mitochondrial study ever published. So the biggest study ever conducted on an mtDNA substitution rate lands perfectly on the Biblical timeline of 6,000 years using Árnadóttir et al. 2024 results. That means this is probably closer to the true global average rate since the majority of studies seem to obtain results around this range with side branches having much faster rates such as Madrigal et al 2012 in the Atenas, Costa Rica population. While slower rates are found in the Amish population noted by Parsons et al 1997. The empirical picture is therefore not one of a perfectly uniform metronome ticking at an identical rate in every woman, every family, and every population. It is a distribution of rates around a central tendency.

That point matters because a mean and a maximum are different statistical objects. The 14.1 differences reported by Budowle et al. represent a high average pairwise distance within an African population sample. The 32 and 36 difference observations examined elsewhere in this paper are extremes near the upper tail of the observed diversity distribution. There is no statistical requirement that the rate which best reproduces the center of a distribution must also pass exactly through its most extreme observation. Indeed, if mutation and substitution are stochastic processes, variation around the expected value is unavoidable even under a completely constant underlying rate. This is not merely a statistical convenience. The deepest pairwise comparisons are concentrated on precisely the lineages carrying the control region's known mutational hotspots, such as position 16189, which Santos et al. (2005) observed mutating rapidly and independently in two separate families, and elevated-heteroplasmy lineages of the same kind in Wei et al. (2019) and Peng et al. (2025) recovered at low detection thresholds; the same fast sites that inflate a lineage's substitution count are what place it in the upper tail of the diversity distribution, so applying an upper-tail rate to an upper-tail observation matches like with like rather than switching rates opportunistically. Once the empirically observed lineage-to-lineage rate heterogeneity is added, the expected spread becomes wider still. Consequently, it is entirely reasonable for an average diversity value to correspond to a rate in the 1/40–1/50 range while exceptionally divergent maternal branches are accommodated by considerably faster rates that have also been directly observed.

This provides an important interpretation of the value used elsewhere in this paper. We do not need to maintain that 1/19.947 represents the universal mutation clock of humanity. Its importance is different. It demonstrates that when multiple directly observed, HVR-compatible pedigree results are pooled, the resulting empirical signal is sufficiently rapid to generate even the deepest modern-human control-region differences within the proposed recent chronology. It therefore functions as a high-diversity stress test. The calculation asks whether the upper end of existing human diversity is too great to have arisen in several thousand years when compared with rates actually observed in pedigrees. Our answer is no. The observed maximum remains within the range that measured pedigree rates are capable of generating.

Conversely, when the question changes from the maximum amount of diversity to the typical amount of diversity, slower measured rates become more relevant. This is exactly what the *Budowle* comparison demonstrates. We do not have to force the fastest pedigree result onto the average African American sequence, nor do we have to force the slowest pedigree result onto the most divergent pair. The observed pedigree literature already contains both. The central and tail portions of the modern diversity distribution can therefore be compared with corresponding central and high-rate portions of the empirical pedigree distribution. In our view, this is biologically more realistic than insisting that every maternal lineage in human history must have followed one immutable clock.

Some may criticize that an added HVR2 unknown rate was added to (Madrigal et al 2012), however it was only added for calculations that were missing yet required for that single study. If we focused only on HVR1 for this study, we would be much more limited but can still even obtain similar results. There is little difference at all in the end and we will show you that directly here. Since they reported a rate nearly identical to *Parsons* but slightly faster, we should expect a date for mitochondria Eve before *Parsons* reported 6,500 years, and that is exactly what we find even using these differences found in HVR1. To test this, critic Stefan Frello posted that the max nucleotide substitution difference between two humans in the the HVR1 region is 12 (gene bank KJ669140 and HM771221).

Madrigal et al. (2012) reported multiple HVR1 mutation-rate estimates, including slower estimates and an average which is found in the abstract. Their upper estimate incorporates seven mutations across 273 meioses, yielding a rate of $7/273 = 0.02564$ mutations per HVR1 sequence per generation, or one mutation every 39 generations. Across the 360-base-pair region, the authors report 71.1×10^{-6} mutations per site per generation. Using their empirically measured female generation interval of 28.3 years, they report an annual rate of 2.51×10^{-6} mutations per site.

Taking a pairwise HVR1 distance of 12 nucleotide differences as the input, a simple constant-rate model estimates the common-ancestor time by dividing this distance by the combined mutation rate of two descendant lineages. Using the published annual rate gives $T = 12/[2 \times 360 \times (2.51 \times 10^{-6})] = 6,634$ years. Calculating directly from the unrounded pedigree frequency gives $T = (12/2) \times 39 \times 28.3 = 6,622$ years; the small difference reflects rounding of the published rate. Applying instead the 20 year generation interval used by *Parsons* et al. (1997), while retaining *Madrigal's* per-generation mutation frequency, gives $T = (12/2) \times 39 \times 20 = 4,680$ years. This is exactly what the data shows and how yet again just letting the data speak for itself, confirms the biblical timeframe.

Even Mumm et al. (1997) at approximately 1/59 generations, bracket the approximately 1/47 generation rate required for a 12 substitution pairwise HVR1 maximum to accumulate in 5,650 years. So even using HVR1 published rates only, we can still easily explain the high diversity found within the human population today. We just decided to be much more thorough and incorporate as many studies and as much data as possible for a more complete meta-analysis. Plus, Mumm's rate is near the average of the spectrum for HVR1, even *Madrigal's* HVR1 rate is a bit slow and closer to the average rather than the high end. We like their study because the pedigree's themselves tested were all very deep rooted and not shallow dyad or triad. This argument shuts down critics who dismiss *Parsons* rate being fast only because its mostly single generation pedigrees.

People are under this strange impression that if a study doesn't land directly on the biblical timeline, then it refutes it or even that every study needs to land exactly on the 6,000 or 7,600 year biblical creation event. Let's clear this misconception up.

Most studies should actually obtain results much later than the 6,000 to 7,600 year biblical creation timeline. The reason why is that the average diversity is what is common and only explained with a slower average mutation rate, not a faster one. The fastest rates are only used to explain the highest diversity, so therefore the average rate is used to explain the average diversity.

What is the average diversity? 10.6 +/-4.9, a range 5.7 – 14.1.

Populations	# Matches/ # Comparisons	Freq ($\times 10^{-3}$)	Avg. # Different Nucleotides $\pm$ SD	(Range)
<i>Within group</i>				
Pairwise Comparisons				
African American	24/11026	2.18	14.1 $\pm$ 5.8	(0–32)
African (Sierra Leone)	61/6105	9.99	13.1 $\pm$ 5.6	(0–31)
US Caucasian	669/182106	3.67	7.9 $\pm$ 3.7	(0–30)
Austrian Caucasian	15/5050	2.97	8.4 $\pm$ 4.1	(0–26)
French Caucasian	23/5886	3.91	7.2 $\pm$ 3.4	(0–19)
Hispanic	36/4851	7.42	12.6 $\pm$ 5.7	(0–30)
Asian American	13/1653	7.86	10.9 $\pm$ 4.0	(0–24)
Japanese	52/13041	3.99	9.7 $\pm$ 3.9	(0–23)
All Eight Databases	1559/969528	1.61	10.6 $\pm$ 4.9	(0–36)

Figure 12. From Budowle et al 1999 Table 3.

That means the average substitution rate for HVR1 & HVR2 should fall between one substitution every 42.6 generations to one substitution every 133 generations (1/42.6 – 1/133) depending on the date of creation. With a mean expectation of around one substitution every 57-72 generations for 10.6 differences. For 6,000 years the range would be one substitution every 42.6 – 105.3 generations for and for 7,600 years 53.9 – 133.3.

For the low 5.7 average differences:

- at 6,000 years: 1 substitution every 105 generations
- at 7,600 years: 1 every 133 generations

For the high 14.1 average differences:

- at 6,000 years: 1 every 42.6 generations
- at 7,600 years: 1 every 53.9 generations

So the total rate range needed to encompass both observed diversity values over your 6,000 – 7,600 year chronology is approximately: 1 HVR-I + HVR-II substitution every 43 – 133 generations per maternal lineage.

So the central required rate is approximately: **1 substitution every 64 generations**, with an expected range of about 1/57 – 1/72 across the 6,000 – 7,600-year chronology.

This range explains the common range of diversity we see in populations today and therefore represents rates that should be observable in pedigree studies if our timeline is true. It should also be reflected in a pooled meta-analysis of pedigree mutation-rate studies, which we will examine next.

But first, what happens when we take these rates and calculate a date for the most recent common ancestor?

Using 1 substitution every 62 generations per lineage, a 20-year generation time, and a maximum HVR-I + HVR-II pairwise diversity of 39 differences: $19.5 \times 62 \times 20 = \mathbf{24,180 \text{ years}}$

So using the average substitution rate the estimated MRCA is approximately 24,180 years ago using the maximum observed diversity.

Notice how far this MRCA estimate falls outside the 6,000 – 7,600-year timeframe. Yet this does not invalidate the model, because the same substitution rate still successfully explains the average level of HVR-I + HVR-II diversity observed within that biblical timeframe. What looks like a failure is actually a strength. The apparent discrepancy arises only when an average rate is applied to the maximum pairwise diversity, as though the most divergent pair of sequences should be expected to accumulate substitutions at the population-average rate.

That is precisely why the fastest empirically observed pedigree rates are most relevant when evaluating the upper extreme of modern diversity. The average rate should be used to explain average diversity, whereas the highest observed diversity requires comparison with the faster end of the measured rate distribution. Likewise, an average substitution rate should not be treated as the appropriate rate for deriving an MRCA from the single most divergent pair in the population.

This distinction is critical. A slower rate can produce an MRCA estimate well outside the biblical chronology while still correctly reproducing the ordinary diversity expected within that chronology. There is no contradiction in those two results. They are answering two different questions.

When the rates are matched to the diversity they are actually capable of explaining, average rates with average diversity and faster rates with the extreme upper tail the pedigree data become internally consistent with the proposed timeline rather than contradictory to it. The important question is therefore not whether every substitution rate produces the same MRCA date, but whether the observed spectrum of pedigree rates is capable of generating the observed spectrum of modern human mitochondrial diversity within the proposed timeframe.

The conclusion is difficult to escape for critics: modern human mitochondrial diversity is not inherently too great for a recent human origin. The diversity observed in living populations is reproduced by the slower, central range of pedigree substitution rates, while the extreme upper tail of that diversity is reachable by the faster rates that have also been measured directly in human families. No hypothetical acceleration is required, no unobserved mechanism must be invented, and no single universal molecular clock must be imposed on every maternal lineage.

The very spectrum of rates required by the proposed biblical chronology is already present in the empirical pedigree literature and has been for over three decades. The challenge for a recent-origin model was always straightforward: can several thousand years of directly observed human mitochondrial change plausibly generate the diversity that exists today? When the full distribution is compared like with like, average with average, extreme with extreme, the answer from the pedigree data is yes.

Our model has always been easy to falsify in this regard, yet as time goes on the data only confirms it more.

12.1 Testing the global mean

The largest study we have is in an Icelandic population by *Árnadóttir's 2024*, and their results answer a different and narrower question: *How well does their rate of HVR change match to a global mean?* At the population level, an African American high mean pairwise diversity of 14.1 can be generated within 6,000 years by a rate of approximately 1/42.6 and within 7,600 years by approximately 1/53.9. Both required values have close analogues in published pedigree work. At the opposite end of the distribution, the deepest observed modern-human comparisons can be accommodated by the faster pedigree estimates documented throughout this paper. Between those endpoints lies a broad collection of studies reporting intermediate rates. The best way to put this to the test is through a meta-analysis.

A few meta-analyses have been undertaken over the years, but all of them fall before the year 2003, back when few total studies existed. So we will now run our own meta-analyses using old and current studies and see if the mean does fall between one substitution every 62 generations. To obtain a broader empirical estimate of the mitochondrial control-region pedigree rate, a transmission-weighted analysis was performed using pedigree datasets for which observed mutational events and maternal transmission counts could be expressed directly.

The studies considered here include:

- **Defense Science Board/AFDIL (1995):** preliminary observations across approximately 300 generations suggested roughly **1 substitution per 50 generations**.
- **Bendall et al. (1996):** 4 mutations / 360 transmissions = **1/90** in HVR-I.
- **Howell et al. (1996):** 2 mutations / 88 transmissions = **1/44**.
- **Parsons et al. (1997):** 10 mutations / 327 transmissions = **1/32.7** across HVR-I + HVR-II.
- **Mumm et al. (1997):** 1 mutation / 59 transmissions = **1/59** in HVR-I.
- **Soodyall et al. (1997):** 0 mutations / 108 transmissions across HVR-I + HVR-II.
- **Hühne et al. (1998):** 6 heteroplasmic point mutations across 77 mother–child comparisons $\approx$ 1/12.8; some comparisons included HVR-I alone and others HVR-I + HVR-II.
- **Cavelier et al. (2000):** 0 mutations / 292 transmissions across HVR-I + HVR-II.
- **Heyer et al. (2001):** 4 mutations / 508 transmissions = **1/127** across HVR-I + HVR-II.
- **Brandstätter et al. (2004):** 3 newly arising point mutations / 135 transmissions = **1/45** across the control region. A broader definition that also counts two maternal heteroplasmies resolving to homoplasmies would yield **5/135 = 1/27**.
- **Santos et al. (2005):** 11 observed HVR-I + HVR-II changes / 321 transmissions = **1/29.2** using the raw event count; their more restrictive germline subset was **6/321 = 1/53.5**.
- **Madrigal et al. (2012):** maximum HVR-I estimate 7 mutations / 273 transmissions = **1/39**; after removing questionable pedigree branches and a mutational hotspot, the conservative estimate fell to **2/220 = 1/110**.
- **Árnadóttir et al. (2024):** the largest pedigree investigation to date examined **116,663 mother–child transmissions and detected 8,199 mutations genome-wide**. Applying its position-specific HVR rates to the combined HVR-I + HVR-II interval gives an approximate combined rate of **$\sim$ 1/42 - 43 generations**; this is a derived HVR estimate rather than 8,199/116,663, which represents the entire mitochondrial genome.

Using Bendall, Howell, Parsons, Mumm, Soodyall, Hühne, Cavelier, Heyer, Brandstätter, Santos, and Madrigal gives a total of 48 observed mutations across 2,548 maternal transmissions, corresponding to a transmission-weighted rate of: 1 substitution every 53.08 generations (1/53).

The pooled value changes depending on how strictly a “mutation” is defined. Replacing the raw Santos result of 11/321 with its restrictive 6/321 germline subset, and replacing Madrigal's maximum 7/273 estimate with its conservative 2/220 estimate, reduces the pool to 38 mutations across 2,495 transmissions, or approximately:

1 mutation every 65.66 generations (1/66).

This gives a useful conservative sensitivity bound: the primary raw-event pool yields approximately **1/53 generations**, whereas substantially stricter mutation-screening criteria yield approximately **1/66 generations**.

Within all this data we do not encounter a missing rate that must be hypothesized solely to make the chronology work. The rates required by both the center and the upper tail of observed diversity already occur within the empirical human pedigree literature. Seen this way, the apparently wide spread in published pedigree estimates is not a weakness of the argument but a biological feature that should be incorporated into it. Consequently, some families will, by chance or biology, produce several observable changes over a short interval while others produce very few.

This observation also prevents an unnecessary false choice. One does not have to decide that either Árnadóttir's average value is correct or Moreau's rapid rate is correct or Parsons' slow Amish rate is correct. All three can be genuine rate observations of a heterogeneous biological process.

The more appropriate question for historical reconstruction is *“Does the empirically observed distribution of rates have sufficient capacity to generate the distribution of diversity observed in humans today?”* Within this very study you have that answer, and that answer is **yes**.

Our expectations and future predictions are that increasingly large pedigree datasets will narrow the uncertainty around the center of the distribution while continuing to identify unusually rapid and unusually slow maternal lineages at its tails. If our interpretation is correct, future very large, multigenerational datasets using common D-Loop and HVR boundaries and standardized heteroplasmy thresholds should not converge upon the 1/19.947 value simply because it is the pooled upper end value used in this paper. Instead, they will produce a slower population-wide central estimate similar to Árnadóttir's rate, while still retaining a substantial high-rate tail in rare studies. Such results would strengthen our paper, model, predictions and conclusions: slower central rates would account naturally for ordinary pairwise diversity, while the empirically retained high-rate tail would remain available to account for unusually divergent branches. What would challenge the model would be a sufficiently large and representative body of pedigree evidence showing that the rates required by the recent chronology, both central and high-end, do not actually occur.

Other such mutation rate predictions have already been made by Dr. Jeanson, who predicted that the most divergent African populations would eventually be found to have a faster directly measured mtDNA mutation rate, roughly 0.2 – 0.3 whole mtDNA mutations per generation. This prediction is in his 2015 work he calls the stamp of civilization. Later, in *Replacing Darwin*, he made the prediction even more explicit, saying that the most divergent African groups should show something on the order of one mtDNA mutation every 5 – 8 generations, or faster, and that he would not be surprised if some African lineages were approximately twice as fast as the non-African lineages

We have already observed many multi-generation pedigrees that have a rate far faster than the Árnadóttir's average rate. Existing pedigree evidence already demonstrates substantial variation around the Árnadóttir et al estimate, including considerably faster multigenerational observations. These published pedigree empirical rates have remained unfalsified for three decades giving us all the evidence we need as biblical creationists to explain the diversity for a young timeline. Since the goal of any model of ancestry is to explain the diversity via their model, we satisfy that and we do it with observable, testable, repeatable evidence over decades of time in multiple independent studies. The only problem lies on the evolutionary timeline which cannot slow the rate down far enough to reach their ever changing dates that just get older and not younger, compounding the already unresolvable problem.

Our interpretation of the data is therefore more nuanced than assigning one number to the mitochondrial clock. The 1/19.947 pooled estimate is a descriptive upper mean of the selected high end pedigree studies and a useful high-rate test parameter for examining only the upper limits of maximum diversity; it should not be used as or argued again as the definitive rate or global human average rate. A more central rate is appreciably slower, and the average rate of which lands on a 6,000-year timeline is approximately 1/42 which just so happens to coincide with Árnadóttir's 2024 rate and the 7,600-year timeline with a rate of approximately 1/54. These rates suggest a plausible region mean in which such a long-run population average should fall based on our model and timeline. Both faster and slower lineages can coexist around that center but should be more rare the farther out you get from that range (1/42 – 1/54).

That, in our view, is one of the more important implications of the present survey. Human mitochondrial variation does not require us to choose between a single slow clock or a single fast clock. The pedigree record shows a spectrum. Average human diversity is compatible with the slower portion of that directly observed spectrum, while maximum human diversity is compatible with its faster portion. Both ends are empirical. Neither requires the invention of an otherwise unobserved rate like the evolutionary model requires. Whatever final global mean future pedigree research establishes, the striking observation remains that the range of rates already measured within human families is broad enough to encompass the amount of mitochondrial diversity actually present in living humanity within the recent chronological framework tested here.

12. Conclusion

Pooling the HVR-compatible pedigree studies based on fixed, consensus-level differences yields a pooled high-end mean of one substitution every 19.947 generations per maternal lineage. This represents an upper-portion summary of the observed pedigree distribution rather than its fastest individual rate. Applied across the candidate biblical interval of approximately 225 to 380 generations, it predicts approximately 22.6 to 38.1 pairwise HVR-I/HVR-II substitutions between two descendant lineages. The representative deep comparison of 32 differences and the single most extreme comparison of 36 differences both fall within this predicted range. At a 20-year generation interval, reading these distances back through the same clock produces maximum-likelihood coalescence estimates of approximately 6,383 and 7,181 years, respectively, both within the 7,600-year upper chronological boundary tested here.

Importantly, the pedigree-rate inputs were derived independently of the biblical chronology. The chronology supplies the interval over which the rate is tested; it does not determine the rate itself. The analysis therefore proceeds in both directions. Projected forward, the measured pedigree rate predicts a range of mitochondrial diversity that encompasses the deepest observed values. Read backward, those same observed values yield coalescence estimates within the recent interval. The agreement is therefore not limited to converting a selected genetic distance into a desired age: the model also predicts, in advance of that reverse calculation, the approximate amount of diversity expected to arise within its proposed chronology.

Long-term phylogenetic rates are always substantially slower than the rates observed in pedigrees because they are calibrated against independently assigned divergence times dated by radiometric dating. To make the observable genetic data align with these deep time dates, evolution theory invokes selection, lineage loss, finite-sites recurrent mutation saturation, demographic history, and time-dependent rate behavior. The relevant question is not whether such processes exist, but whether their demonstrated magnitude is sufficient to reduce the heritable pedigree signal over the approximately 300 to 380 generations most relevant to the present model and test. On the calculations presented here, all such effects remain limited across this interval, while no quantitatively demonstrated rate-reduction mechanism examined in the study is large enough to move the observed control-region diversity outside the recent-origin range. The rate discrepancy paradox under a deep chronology requires no comparable reconciliation within the shorter chronology examined here. The genetic evidence begs the question. If genetic evidence from observable studies down the line keeps confirming that the radiometric dating phylogenetic evidence is inaccurate, which are we to believe? Is radiometric dating wrong or is the genetic data wrong? What if both can be correct at the same time? This is where our model shines and how we can explain the data while evolution is still trying to solve the paradox.

The mitochondrial result also does not stand alone. Evidence for the recent expansion of much rare human variation, including the population-expansion estimates discussed from Tennesen et al. (2012) and Fu et al. (2013), falls within the same narrow recent interval. The modest average growth required to reach the present population from a small founding group, the comparatively recent genealogical convergence of living humans, the rapid appearance of the documented historical record, and the ancient-DNA preservation evidence examined

here all provide independent additional lines of consilience. Their significance is that several distinct empirical pathways match the same recent founding-and-bottleneck framework. Of these, the mitochondrial pedigree clock and ancient-DNA decay provide the most direct chronological tests, while the demographic, genealogical, and historical observations offer corroborating context.

The recent-origin interpretation is also falsifiable. If future studies obtain higher levels of diversity between different demographics of people that cannot be reached by observed pedigree substitution rates observed in pedigree studies then our model at being able to explain the diversity within the biblical timeframe is falsified. Also, if progressively better-resolved maternal pedigrees could reveal a new mechanism of systematic decline in the heritable hypervariable region substitution rate over the relevant few hundred generations and transmission models could show that substantially more mutations are concealed than the present analysis indicates would also falsify the model. So if future data systematically move the pedigree clock away from the range documented here, the recent-origin model is falsified. If increasingly large and multigenerational datasets continue to reproduce rates of the same general magnitude while observed diversity remains within the model-implied range, the case for a recent mitochondrial chronology will become even stronger and confirmed while at the same time more difficult to dismiss.

This study therefore moves the creationist mitochondrial argument beyond a general appeal to rapid pedigree rates. It presents a specific empirical rate, applies that rate to a defined chronology, calculates the diversity expected under the model, compares that prediction with observed human variation, quantifies the principal objections, and specifies conditions under which the interpretation can fail. The remaining disagreement is consequently quantitative: what empirically demonstrated mechanism, operating on the relevant sequence region and over the few hundred generations examined here, reduces the directly observed heritable rate enough to overturn the result?

The proverbial ball, therefore, is no longer in the creationists' court simply to demonstrate that a recent chronology can be made compatible with genetics. This study has stated its clock, its chronology, its predicted diversity range, and its falsification conditions. The conventional framework must now demonstrate, by measurement, why those directly observed rates should not and cannot be read over the timescale actually under examination. The recent-origin model has stated its clock, its prediction, and its conditions for failure; the conventional framework must now answer with evidence against it.

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GenBank and sequence-data records

National Center for Biotechnology Information. (n.d.). Homo sp. Altai complete mitochondrial genome sequence from Denisova Cave (GenBank accession No. FN673705) [Data set]. <https://www.ncbi.nlm.nih.gov/nuccore/FN673705>
The Neanderthal panel consisted of GenBank accessions FM865407 (Feldhofer 1), FM865408 (Feldhofer 2), FM865409 (Sidrón 1253), FM865410 (Vindija 33.25), and FM865411 (Mezmaiskaya 1), the five complete Neanderthal mitochondrial genomes of Briggs et al. (2009). The Denisovan panel consisted of FN673705, FR695060, KT780370, and KX663333.

Methodological and data-availability notes

Neanderthal and Denisovan control-region consensus sequences were generated independently from complete mitochondrial-genome records obtained from GenBank. The Neanderthal panel consisted of FM865407, FM865408, FM865409, FM865410, and FM865411, while the Denisovan panel consisted of FN673705, FR695060, KT780370, and KX663333. Complete mitochondrial sequences were aligned to the revised Cambridge Reference Sequence, NC_012920.1, to preserve rCRS coordinate numbering. A majority-rule consensus was assigned at each callable reference position separately for Neanderthals and Denisovans. Tied or unresolved positions were coded as ambiguous or missing. Insertions and deletions were recorded separately and excluded from substitution counts. HVR1 and HVR2 were analyzed separately and, where combined, concatenated as a 610-bp panel consistent with the HVR1–HVR2 window used throughout this paper.

The human consensus sequence was the Reconstructed Sapiens Reference Sequence described by Behar et al. (2012), distributed in FASTA format by PhyloTree and reconstructed from the revised Cambridge Reference Sequence, NC_012920.1, with the documented RSRS-defining substitutions applied.