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Robert Carter
Creation Ministries International

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GENEALOGICAL VS PHYLOGENETIC MUTATION RATES: ANSWERING A CHALLENGE

Robert W. Carter, Creation Ministries International, P.O. Box 350, Powder Springs, GA 30127 USA r.carter@creation.com

ABSTRACT

There is a discrepancy between the mutation rate we can measure today and the rate at which evolution is supposed to have proceeded. The former is sometimes called the genealogical mutation rate, for it is obtained by comparing individuals whom we know to be related. The latter is sometimes called the phylogenetic mutation rate. It is generally calculated by counting the fixed differences between two species and dividing by the estimated time since their common ancestor. Genealogical mutation rates are several orders of magnitude faster than phylogenetic estimates. This causes problems for the evolutionary model. For example, using the genealogical method would place Y Chromosome Adam and Mitochondrial Eve well within the biblical time frame. The evolutionary community often uses appeals to natural selection, genetic drift, or theoretically low mutation rates to explain away the discrepancy. In this study, the population modeling software *Mendel's Accountant* and simple statistics were used to show that these explanations do not work. The genealogical mutation rate is, in fact, a serious challenge to evolutionary theory.

KEY WORDS

phylogeny, genealogy, mutation, Y Chromosome Adam, Mitochondrial Eve, natural selection, genetic drift

I. INTRODUCTION

The fact that genealogical estimates yield much faster mutation rates than phylogenetic estimates has been known for many years (Wieland 1998, 2006). Evolutionists generally claim the dilemma is irrelevant or easily solved (c.f., Carter 2021), for a variety of reasons which will be explained below. Even so, the issue continues to pop up in evolutionary writing (e.g., Connell et al. 2022). When estimating things like the time to a most recent common ancestor (TMRCA), they almost universally use the phylogenetic mutation rate. This is obtained by dividing the number of differences separating two species by the assumed time since their last common ancestor (Mishmar et al. 2003).

At least, this is what is generally presented. Occasionally, a study will use known archaeological points to fix the tree in time. The dating of such events is generally based on radiometric methods. This has been attempted for mitochondria (Friedlaender et al. 2005) as well as Y chromosomes. In fact, the most significant Y chromosome studies used the peopling of the Americas as a fixed reference point, calling it a "sanity check". This exact phrase was used in the main paper that contributed the Y chromosome data to the 1000 Genomes Project (Poznik et al. 2016), as well as in an earlier paper by a different group of authors (Behar et al. 2012). While discussing this, Carter et al. (2018) stated, "Clearly, they are prepared to reject measured mutation rates in favor of evolutionary assumptions if the measured rates turn out to be too high."

Phylogenetic methods (whether based on archaeology, radiometric dating, or something else) generally yield mitochondrial mutation rates on the order of 10^{-8} mutations per site per year. Given a 16,569-nt genome and a ~30-yr generation time, that amounts to approximately 1 mitochondrial mutation every 200 generations. This is not actually the 'mutation' rate. Instead, it is the substitution rate, or the rate at which new mutations replace the original genetic variant across the entire population. On the other hand, the genealogical mutation rate is a better estimate of the real-time mutation rate (after subtracting lethal mutations).

The phylogenetic mutation rate is clearly influenced by evolutionary assumptions, but the genealogical rate is not completely free of them either. Since the error rate in large sequencing databases is on the same order of magnitude as the expected mutation rate, the data must be highly filtered before any mutation rate estimates can be made. Earlier genetic databases had so many errors that much of the data were unusable (Carter et al. 2008), but quality control has improved. Still, the intrinsic error rate complicates all calculations. Among creationists, Jeanson has done the most work on this (see Jeanson and Holland 2020) and we are indebted to the detailed analyses of this subject that he pioneered. During data filtering, it is highly likely that many real mutations are removed, which would lower the genealogical mutation rate. Even so, the rates are still far too high, meaning that Mitochondrial Eve and Y Chromosome Adam would be placed too recently in time for the evolutionary model.

In their study on the mitochondrial mutation rate in *Daphnia pulex*, Xu et al. (2012) said:

> "Despite the great utility of mitochondrial DNA (mtDNA) sequence data in population genetics and phylogenetics, key parameters describing the process of mitochondrial mutation (e.g., the rate and spectrum of mutational change) are based on few direct estimates. Furthermore, the variation in the mtDNA mutation process within species or between

> lineages with contrasting reproductive strategies remains poorly understood."

One would think that something of this magnitude would be well studied by now. Yet, it is not like we have no information on the topic. Indeed, much work has been done both on the short-term mutation rate and the variability in mutation rates.

Genealogical studies have consistently produced rates much faster than phylogenetic studies. Hardouin and Tautz (2013) determined that the measurable fixation rate both in mice that had colonized the remote Kerguelen Archipelago and in lab-cultured mouse strains is about six times higher than that of phylogenetic estimates. Madrigal et al. (2009) measured a mitochondrial mutation rate of approximately 0.5 per generation in deep-rooting family trees. They also summarized prior studies, which claimed mutation rates ranging from 0.12 to 1.2 per generation (excluding the one study that discovered zero mutations among 292 generational steps). More recently, Connell et al. (2022) published the genealogical rate for 225 individuals from Norfolk Island spanning 345 generational events. Even though their estimate equated to only 0.029 mutations per generation, they claim this was 16 times higher than typical phylogenetic estimates.

Since only a few dozen mutations separate most human mitochondrial groups, with a maximum separation of only a hundred or so (Carter et al. 2008), a mitochondrial mutation rate on the order of 0.5 per generation (Madrigal et al. 2012) would place Mitochondrial Eve in the recent past. Assuming an average generation time of about 30 years (Helgason et al. 2003), biblically, if humans have been around for ~6,000 years (Hardy and Carter 2014), only about 200 generations have occurred in human history. The number of mutations seen is approximately what would be expected (e.g., 200 generations x 0.5 mutations/generation = 100 mutations). That, of course, is a very rough estimate.

On the other hand, there is no reason to expect mutation rates to

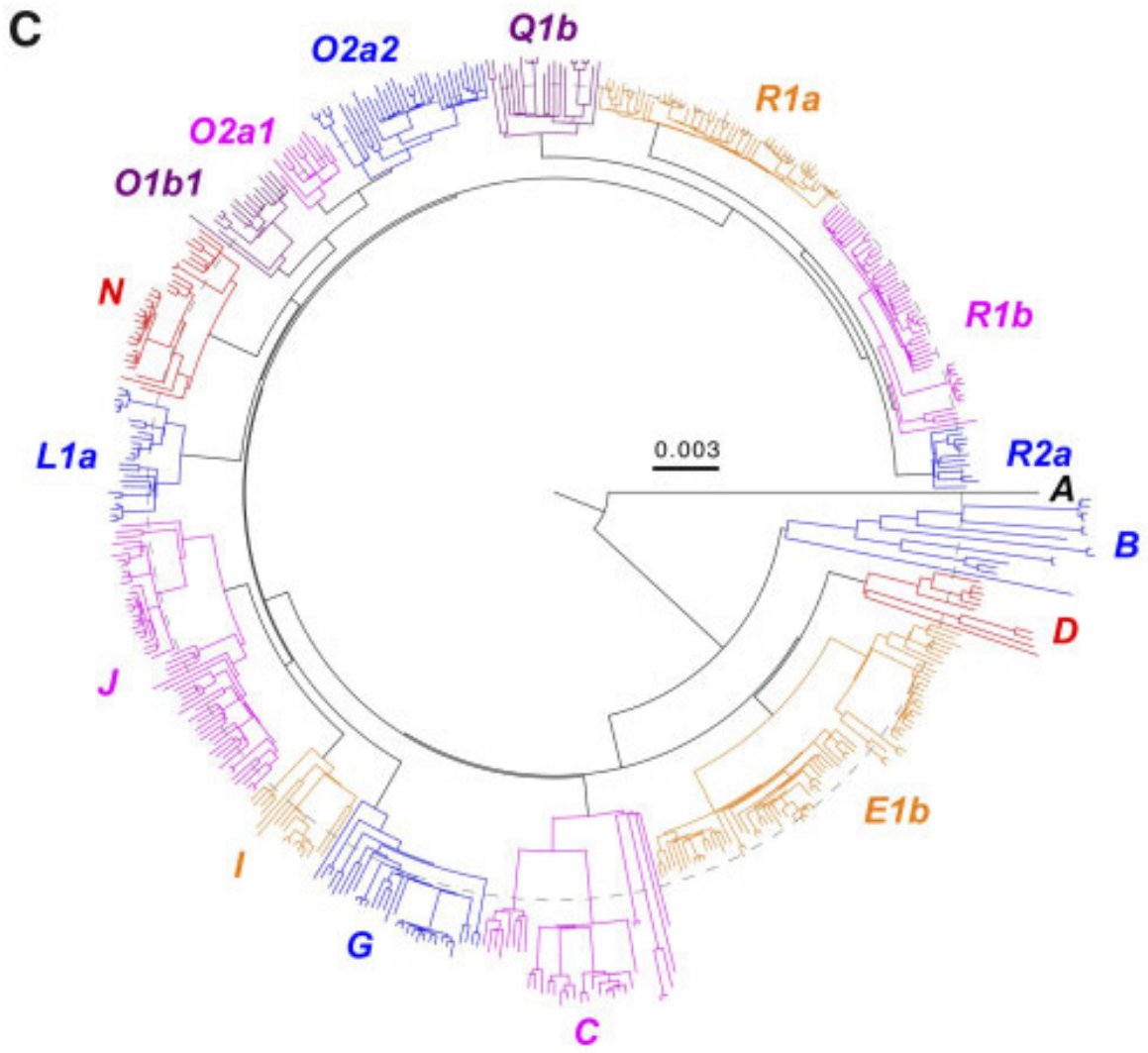

Figure 1. The Y-chromosome family tree according to the Human Genome Diversity Panel (After Ding et al. 2021). The dotted line represents the average divergence from the (presumed, evolutionary) common ancestor of all living males. Since the number of mutations is proportional to branch length, clearly the men from several groups are well above average in the number of mutations they carry (e.g., A and B). One can even see differences in average branch length within the major groups (e.g., D and C). The mutation rate has not been constant across time and geography.

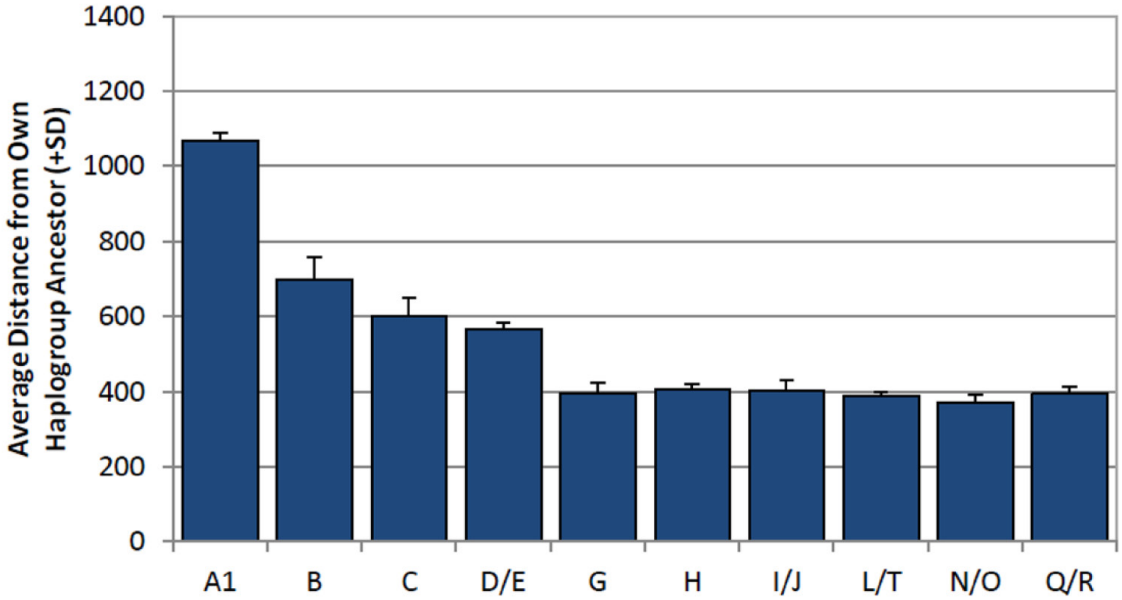

Figure 2. The average distance (+ 1 SD) from each Y chromosome group ancestor to the founding member of the group, after Carter, Lee, and Sanford (2018).

have remained constant across all genealogies, all populations, all time, and all environments (Carter 2021). Claims have been made that identical mitochondria have been found in individuals that lived fully 8,000 years apart (Hublin et al. 2020). This seems preposterous, based on what we know about mitochondrial mutation rates, but they did find two individuals with identical mtDNA who did not live at the same time.

The rate of fixation is also strongly affected by population size (Cabrera 2020). In fact, demographic changes have a much greater effect than selection, and both sides in the creation-evolution debate agree that the human population has undergone dramatic changes in size, both in the long term and locally. It seems logical that the probability of transmission of a new mutation should be similar to the probability of fixation for that mutation. Each measurement approaches 1/(2N) in stable populations. In fluctuating populations, however, the rate of fixation can be proportionally higher (in shrinking populations) or lower (in growing populations). On the other hand, Nabholz et al. (2009) called the mitochondrial molecular clock "erratic". Studying birds, they discovered that the mutation rate in the mitochondrial cytochrome *b* gene was highly variable among different species. They concluded by saying, "Mitochondrial data tell nothing about species population sizes, and strongly depart the molecular clock assumption." Översti and Palo (2022) also demonstrated differential mutation rates among various human mitochondrial lineages.

Likewise, since only a few hundred mutations separate the men on the Y chromosome family tree (Carter et al. 2018), a Y chromosome mutation rate on the order of 1–3 per generation (Jeanson and Holland 2020) would place Y Chromosome Adam only a few hundred generations ago, squarely in the biblical ballpark. While we do not know the actual Y chromosome mutation rate, even low-resolution tandem repeats in Y chromosome data have been used to differentiate father and son in over one quarter of sequenced father-son pairs (Ballantyne et al. 2014). Clearly, the mutation rate is quite high.

Similar to the case for mtDNA, Ding et al. (2021) showed quite clearly that different branches on the Y chromosome family tree have accumulated significantly different numbers of mutations in the same amount of time (Fig. 1). Carter et al. (2018) drew the exact same conclusions a few years earlier (Fig. 2). Ding et al. also showed that the Y chromosome mutation rate in cell cultures derived from the men in their study also comported to the branch lengths on the tree. In other words, intrinsic genetic factors influence the mutation rate. One cannot assume the rate is the same among all men without factoring in the rest of the genome, and we do not yet know how to do that.

There are also questions about where to put the 'root' for phylogenetic calculations. "Using prior knowledge" Poznik *et al*. (2016) placed the founding human Y chromosome at the midpoint between two deeply rooting African Y chromosome lineages. Applying 'sanity checks' and unspecified 'prior knowledge' to critical scientific analyses does not build confidence in the mind of the reader. Even so, the evolutionary mtDNA and Y chromosome roots are still within the range of explanations when using known genealogical mutation rates.

One additional factor that complicates these estimates is something Carter (2019b) called "patriarchal drive". This deals with the excess genetic load very old people would be adding to the population if they had children in the early post-Flood years. In short, the inner branches on the Y chromosome tree (and possibly the mtDNA tree) were created all at once. This reduces the number of generational steps from the middle of the tree (ancestors) to the branch tips (living people). Thus, the genealogical rate was probably faster in the past, so using a modern average rate is a conservative approach that favors evolutionary history.

Natural Selection

There are two main objections evolutionists employ when trying to discount genealogical mutation rates: natural selection and genetic drift. There are problems with each argument. First, there is no

way for natural selection to slow down the rate of genomic change without seriously risking extinction. Reducing reproductive output comes at a cost and there are only so many offspring a population can lose before it collapses (ReMine 2005).

Second, selection only works on alleles that have a significant effect on fitness, and most mutations are expected to be neutral or nearly so. This subject was thoroughly reviewed by Sanford (2014), so it will not be discussed further here. The important thing to understand is that, if most mutations are selectively neutral, it is impossible to create a significant separation between the short-term mutation rate and the long-term accumulation of mutations. There is also a strong interaction between the nuclear and mitochondrial genomes. After discussing the joint mitochondrial-nuclear genotype, and after claiming that the mitochondrial-nuclear interaction is the unit of selection, Dowling et al. (2008) ask, "How much sequence variation in mtDNA is necessary to produce a phenotypic effect?" This number is not yet known, but the mitochondrial genome is not an independent entity. Nuclear effects further complicate the selective arena.

Third, by invoking selection, all mitochondrial molecular clocks could suddenly be wrong. The timing of Mitochondrial Eve is partially based on the assumption of neutrality. If selective mutations occur, the rate of change could speed up (positive selection) or slow down (purifying selection) among the various lineages. Consider that humans live in radically different environments than the supposed source environment (i.e., the forests and plains of equatorial Africa) and there is little reason to believe there are no selective forces at play. Yet, those selective forces must be equivalent among all subpopulations, at all times, and in all places or the molecular clock hypothesis fails. Similar things could be said for the timing of Y Chromosome Adam. Thus, the data from Ding et al. (2021) might be pointing us in a very interesting direction.

Fourth, some skeptics are arguing the wrong thing. Phylogenetic change is the rate at which *species* diverge. Yet, the mutations we are seeing in the mtDNA and Y chromosomes is the rate at which individuals within the population are *diverging from each other*. There is essentially no species-level divergence occurring within modern humans. The population size is too large and we have gone through a thousands-of-years-long exponential growth phase. These factors have prevented the fixation of any new mutations, so there has been no net change within the nuclear or mitochondrial genomes for quite some time. Even so, evolutionary estimates of the substitution rate within the human mitochondrial genome are on the order of one substitution every 2,400 (Rieux et al. 2014) to 3,500 (Soares et al. 2009) years. These dates are constrained by the accuracy of radiometric dating techniques, which are not part of this study, but see Carter (2022a). And yet, the estimated substitution rate for the individuals in the study of Rieux et al. (2014) were much more consistent than the substitution rates determined for the internal branches (e.g., dated demographic events) of a phylogenetic tree that included those individuals. There is a time-dependency to the data, so clearly one cannot assume a constant mutation or fixation rate through all human history.

Population Genetics

And yet, the substitution rate is also often misunderstood. The mathematics of population genetics tells us that the short-term, measurable mutation rate should approximate the phylogenetic rate. According to standard population genetics theory, the fixation rate (also called the substitution rate) of new neutral mutations should be directly proportional to the base mutation rate (μ). This can be found throughout the literature (e.g., Kimura 1983) and is part of any course in population genetics (e.g., the classic population genetics textbook of Hartl and Clark, 1997). The number of new mutations per generation is simply $2N\mu$, where N is the population size and μ is the base mutation rate per individual. The "2" is included for diploid species (e.g., humans). For neutral mutations not affected by selection, the fixation rate is inversely proportional to its frequency in the population, or $1/(2N)$. Again, the "2" is included for diploid species. Multiplying these yields $2N\mu/(2N) = \mu$.

Yet, the rate of substitution also depends on population structure. The time to fixation for new, neutral mutations is approximately 4Ne for diploid systems (e.g., nuclear variants) or 2Ne for haploid systems (e.g., mitochondrial variants). Here, "Ne" refers to the effective population size. The amount of subdivision within a population will affect the overall rate of change. Strongly divided populations behave as if they were smaller, so they have a lower "effective" population size. Yet, this can safely be ignored, as the effect is less than the orders-of-magnitude difference between the genealogical and the phylogenetic mutation rates. Also, the following calculations assume a well-mixed population with random mating (e.g., no population structure), so the modeled population sizes are equivalent to Ne.

Thus, the substitution rate of neutral mutations in the population is equivalent to the initial mutation rate, by necessity. In other words, since most mutations are selectively neutral, genealogical mutation rates should be directly applicable to estimates of the timing of Y Chromosome Adam and Mitochondrial Eve, as well as any autosomal genes. The fact that Y chromosomes and mtDNA are haploid does not matter.

The discussion so far has dealt with theoretical, neutral mutations only, so the question boils down to the rate of non-neutral mutations and the ability of selection to remove them from the population. This can be modeled. One purpose of this study was to provide such a model.

Soares et al. (2009) examined this in detail, but from a purely phylogenetic perspective. After estimating the human-chimp split time, they applied a simple metric (number of mutations/evolutionary time) to generate a phylogenetic mutation rate, assuming in the process that humans and chimps *did* share a common ancestor and that this ancestor lived millions of years ago. They reasoned that newer mutations sit near the tips of the tree and older mutations are the ones shared by many sub branches. By examining the mutation spectra of 'old' vs 'new' mutations, they discovered that there were fewer missense (e.g., amino-acid-changing) mutations in the former. They reasoned that natural selection had removed many deleterious mutations and so the short-term genealogical mutation rate must be adjusted downward when estimating the timing of ancient genetic events.

The rate they calculated was solely based on a presumed common ancestor with chimpanzees at least 6.5 MA. By making this assumption, they were able to estimate the split time for all Old-World mon-

keys (25.2 MA, a group that includes everything from gibbons to humans), human-gorilla (9.4 MA), and chimp-bonobo (2–2.5 MA). Furthermore, they were able to estimate the timing of specific historical events in human history, e.g., the settling of the Canary Islands and Remote Oceania, and the post-Ice Age resettlement of Europe.

It is possible, however, that more missense mutations can be found among newer mutations because genetic systems are breaking down due to the effects of entropy and the Curse. As far as timing the rise of the great ape lineages, all they are doing is uncovering the differences God built into their initial genomes. One of the basic premises of creation thought is that God designed hierarchically (Cserhati and Carter 2020), so some pairs of created groups are necessarily more similar than others and uncovering those differences does nothing to address the creation-evolution debate. Also, archaeological timing estimates can swing wildly. For example, some are arguing that people were in the Americas long before 15 kya. Consider also the claim by Carter et al. (2008) that the mitochondrial sequence data in this era were problematic. Finally, their rate estimate is divorced from any consideration of real-world biological mutation rates. After assuming a human-chimp split time in the millions of years range, their mutation rate was biased downward by several orders of magnitude. For these reasons, and more, attempts to peg archaeology to genetics are clearly fraught with difficulty.

Genetic Drift

A second work-around evolutionists sometimes employ is an appeal to genetic drift. Most new mutations are lost from the population quickly. If the probability of fixation is proportional to allele frequency (f), then the probability of loss is proportional to $1 - f$. Any new mutation, therefore, has a very high likelihood of being lost. Rupe and Sanford (2013) estimated that something like 99.99% of all new mutations that enter a human-like model population are lost. The rate, however, does depend on population size, yet this can also be modeled. If most mutations are lost, does this not indicate that the short-term rate should be much faster than the long-term rate? No, for even if the probability of any one mutation being lost is high, the sheer number of new mutations entering the population guarantees that some will survive.

Consider that you inherited only one-half of the mutations each of your parents carry. You also inherited only one-quarter of those in your grandparents, and one-eighth of those in your great-grandparents. Looking forward in time, mutations disappear quickly. Looking backward, however, one realizes that each individual has two parents. Even though they only passed down one-half of their mutations, 2 x 0.5 = 1. Likewise, 4 x 0.25 = 1 and 8 x 0.125 = 1. Thus, the mutation load of any given individual is identical to the mutation load of prior generations. Since each individual adds more mutations to the population, drift does nothing to slow down the rate of mutation accumulation.

Even so, natural selection should have some effect. It will be removing some alleles and so there should be a difference between the genealogical and phylogenetic mutation rate. The question is, "How much?" What follows is an attempt to quantify the difference between the two mutation rate estimates.

Table 1. Ending fitness in the modeled populations vs the frequency of neutral mutations (first column). Black: population went extinct prior to the 10,000th generation. Gray: population was trending toward extinction but survived to the end of the model run. White: population survived with no fitness loss or the fitness had stabilized.

f(neut)	Pop Size				
	100	500	1000	5000	10000
0.00	0.000	0.000	0.000	0.102	0.115
0.25	0.000	0.000	0.066	0.148	0.165
0.50	0.000	0.064	0.142	0.245	0.261
0.60	0.000	0.103	0.194	0.305	0.322
0.70	0.000	0.177	0.271	0.390	0.399
0.80	0.008	0.274	0.315	0.517	0.528
0.85	0.020	0.373	0.510	0.603	0.610
0.90	0.275	0.529	0.613	0.699	0.707
0.95	0.436	0.752	0.792	0.840	0.842
0.99	0.903	0.940	0.955	0.965	0.966
0.9999	1.000	1.000	1.000	1.000	1.000
1.0	1.000	1.000	1.000	1.000	1.000

II. METHODS

For full-genome analysis, multiple human-like populations were modeled with the online version of *Mendel's Accountant* (see Carter 2019a for a comprehensive assessment of this program). Default parameters were used for most settings, including a 3-billion-bp genome with 989 linkage subunits. The mutation rate was held to 50 per person. Mutation effects were assigned according to a Weibull distribution with a beneficial/deleterious ratio of 0.0001 and a 50/50 ratio of dominant to recessive alleles. Five population sizes (100, 500, 1,000, 5,000 and 10,000) were modeled for 10,000 generations with eleven proportions of neutral alleles (ranging from 0 to 100%). The ending fitness, the number of fixed alleles, the number (or projected number) of generations to population extinction, the percent of mutations retained, and the average number of mutations per individual were tracked and recorded. Due to the high number of runs, each model was run only once. However, initial prototyping showed that repeated model runs produced highly similar results.

Additional *Mendel* runs were performed to estimate the rate of mutation accumulation in mitochondria. Parameters were similar to those listed above, except that a single chromosome with a single linkage block composed of 16,569 nucleotides was used, and the recombination rate and the fraction of recessive mutations was set to zero. One set of models was designed to reproduce the mutation accumulation curves of other studies. Models were run for 100,000 generations using a variety of mutation rates. Another set of models attempted to produce a modern-looking mutation accumulation in a biblical time frame. The models started with 3 individuals, a reproduction rate (prior to selection) of 2, a growth rate of 1.2 (to allow for plenty of selection during the population growth phase), and a maximum population size of 100,000. The models were run for 250 generations with varying mutation rates. All other parameters were as above.

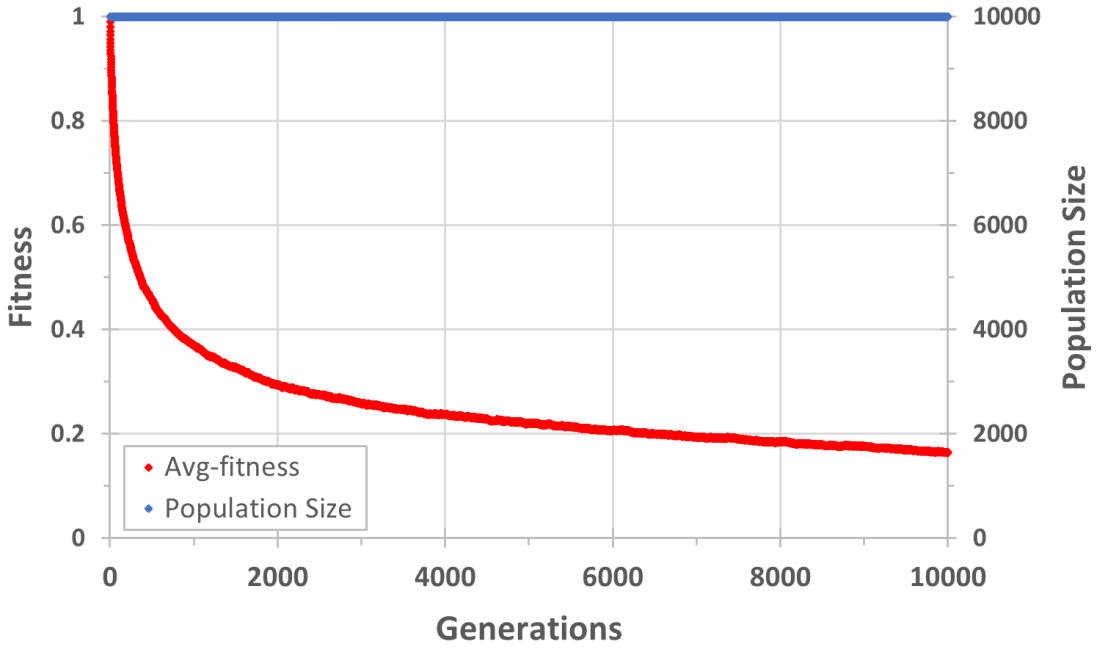

Figure 3. Inexorable loss of fitness in a population of 10,000 individuals with a neutral mutation frequency of 0.25 (yielding approximately 12.5 slightly deleterious mutations per individual per generation).

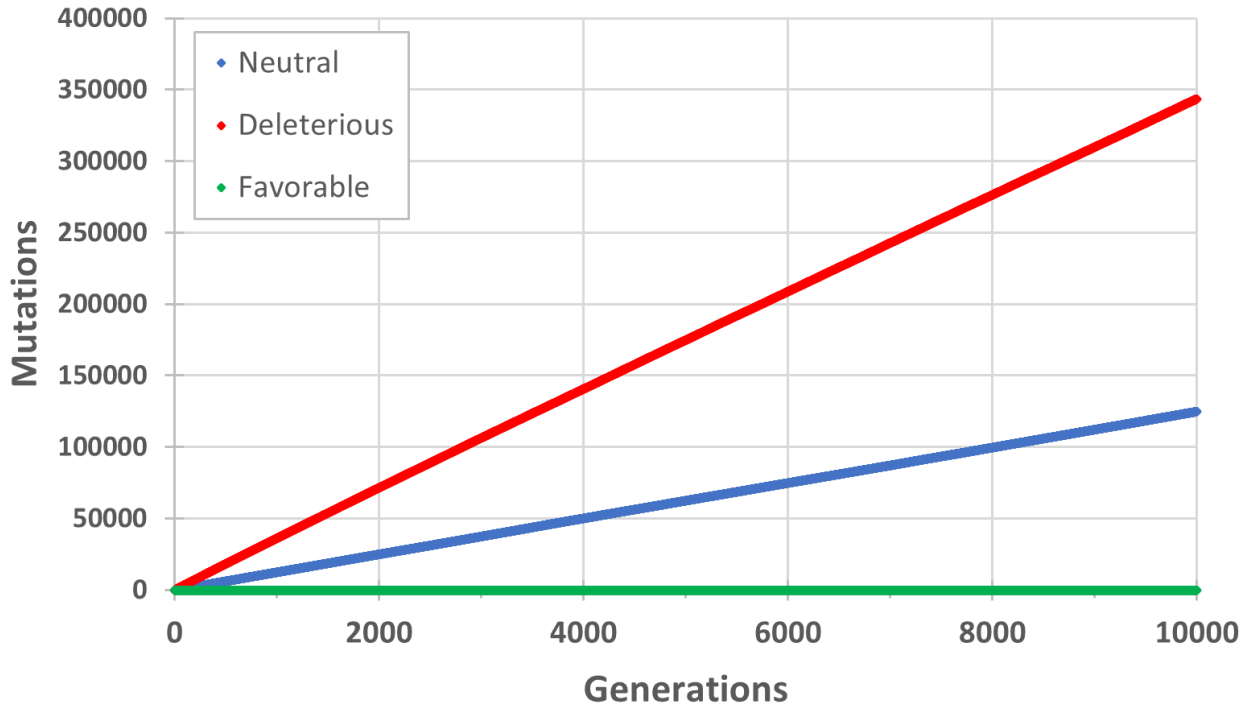

Figure 4. the number of accumulating deleterious (red), neutral (blue), and favorable (green) mutations in a population of 10,000 individuals with a neutral mutation frequency of 0.25. The number of expected neutral alleles (μ x N x generations = 1.25×10^9) matches the number at the end of the run (124,950), given a population-size-dependent retention rate of only 0.01% (c.f., Fig. 6). However, the number of expected deleterious alleles (3.75×10^9) is only slightly larger than the number that remained (343,677), again given a 0.01% retention rate. This tells us that selection cannot remove most deleterious alleles.

A population model modified from that of Carter (2019b) was used to estimate the half-life of neutral mutations in populations ranging from 10 to 100,000 individuals. Each of those models was run 1,000 times. This model was also adapted to track neutral mutations in mitochondria and Y chromosomes. Further modifications were made to include a *Mendel*-like model of probability selection. The main difference is that *Mendel* uses discrete generations, where all parents die and are replaced with their offspring at each iteration. To maintain the population size, surplus individuals are culled according to a probability selection method based on individual fitness scores (the sum of the effects of the specific mutations each individual carries). See Sanford et al. (2007) for additional details. Carter's method uses overlapping generations, so selection had to be on the level of individual survival. To do this, the range of fitness within the population in each year was calculated. During the mortality loop, where individuals are assigned a risk of dying according to an actuarial table, the risk of dying was increased according to the individual's rank within the fitness spectrum. The mutation rate and average mutation effect (controlled by a scaling factor) were adjusted to allow for long-term survival (e.g., 30,000 *Mendel* generations equates to approximately 300,000 years). *Mendel* also has settings for heritability and non-scaling noise. These were effectively treated as "1" and "0", respectively.

III. RESULTS

The ending fitness for each full-genome population model in *Mendel* is presented in Table 1. As expected, small populations with a high rate of non-neutral mutations trended toward extinction. Unexpectedly, some populations lasted for the entire simulation run (10,000 generations, approximately 300,000 model years) yet were clearly *trending* toward extinction the entire time. This included even the largest populations when individuals were receiving more than 5 non-neutral mutations per generation (Fig. 3).

Neutral mutations accumulated in a linear manner (Fig. 4). This was true in all populations. This was also expected, as was the fact that *effectively* neutral mutations behaved as purely neutral ones. Also, the presence of beneficial mutations in the population, which allowed for positive selection, did not slow the accumulation of neutral or deleterious alleles. Even the fixation of strongly beneficial mutations (e.g., selective sweeps) did nothing to slow the rate of mutation accumulation. In the end, the fixation of neutral mutations was directly proportional to the base mutation rate.

In the largest populations, there was no guarantee that *any* alleles would be fixed. In the population with 10,000 individuals, there was no fixation of any neutral alleles in multiple runs. Surprisingly, there were more fixed neutral alleles in the model run with a lower neutral mutation rate. This is due to the removal of individuals with more deleterious alleles, even though the rate of fixation of deleterious mutations was also higher in general.

The total number of mutations that appeared in a model run equals μNg, where g = the number of generations. The percentage of mutations remaining at the end of the run can be obtained from the *Mendel* output files. For purely neutral mutations, the loss of alleles was strong in the smallest populations but increased by two orders of magnitude (from 1.00% retention to 0.01% retention) as N increased

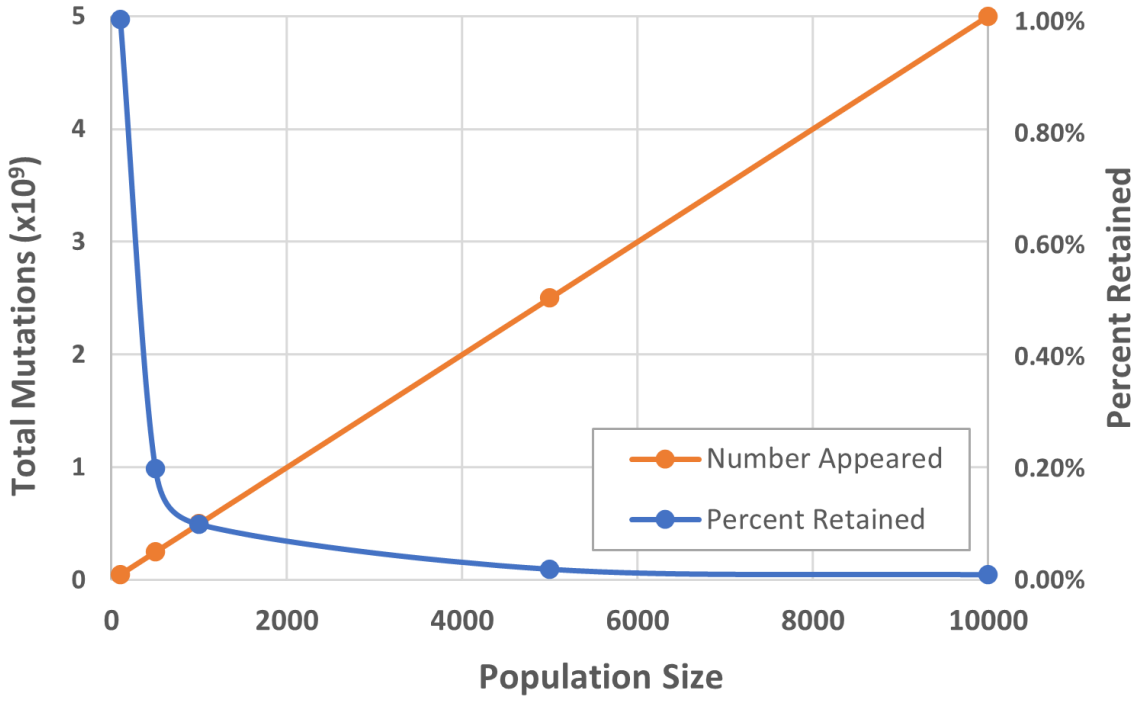

Figure 5. The total number of neutral mutations appearing (orange) and the percent retained (blue) at the end of the model run vs population size. Strong drift (measured here as allele retention, which will eventually translate into allele fixation) is evident in the smallest population, but drift slows to a crawl after the population reaches a few thousand individuals.

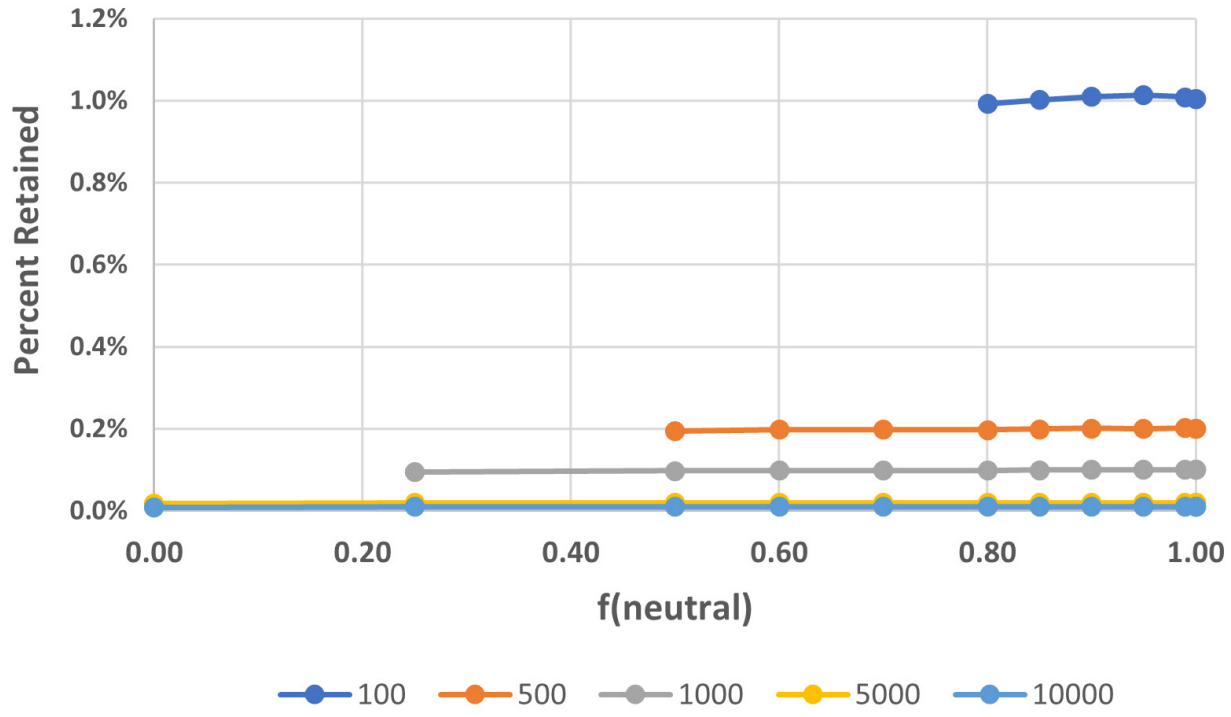

Figure 6. The percent of all mutations retained at the end of each model run vs. the frequency of neutral mutations for each population size. Populations that went extinct are not shown. Even in the largest population there was only an 8.8% difference in allele retention between the model with no neutral alleles (91.4% retention) and the model with all neutral alleles (100.2% retention, with the extra 0.2% coming from the newest alleles that had not yet drifted out of the population).

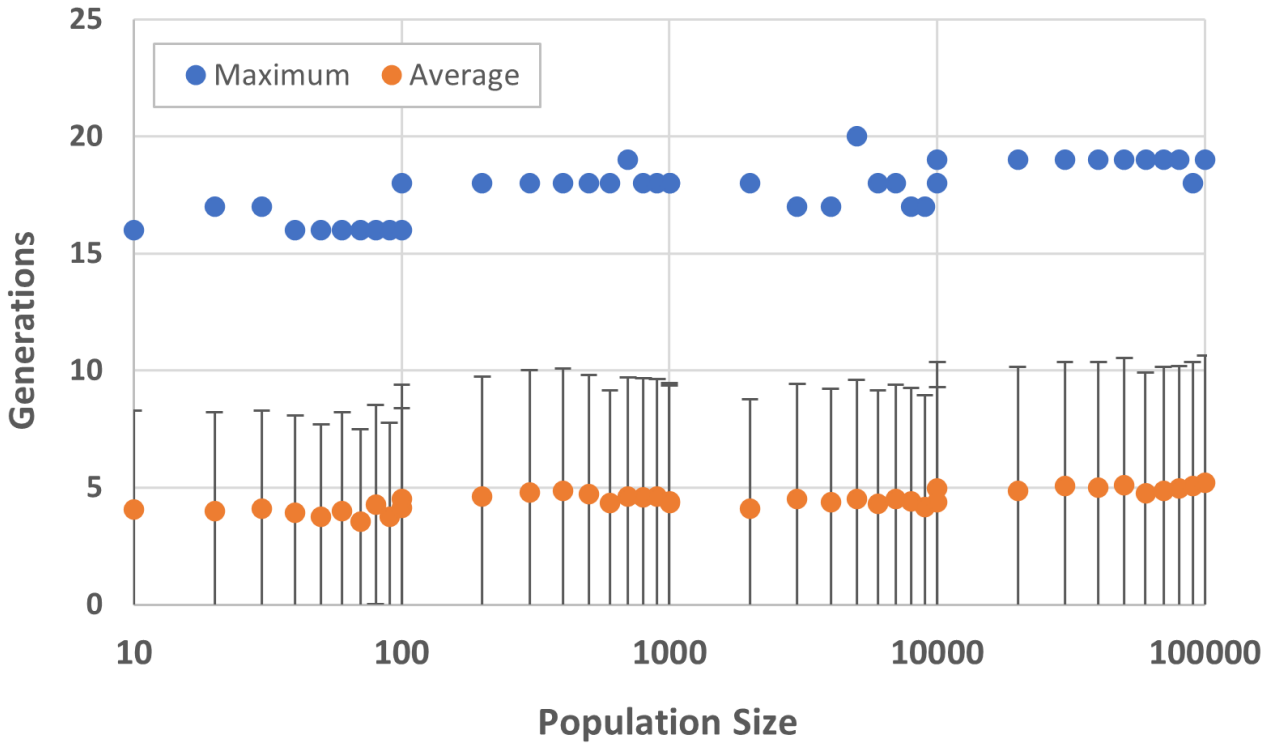

Figure 7. The average (orange) +/- 1 SD and maximum (blue) number of generations before a new neutral allele is lost vs a range of population sizes. Each model was run 1,000 times.

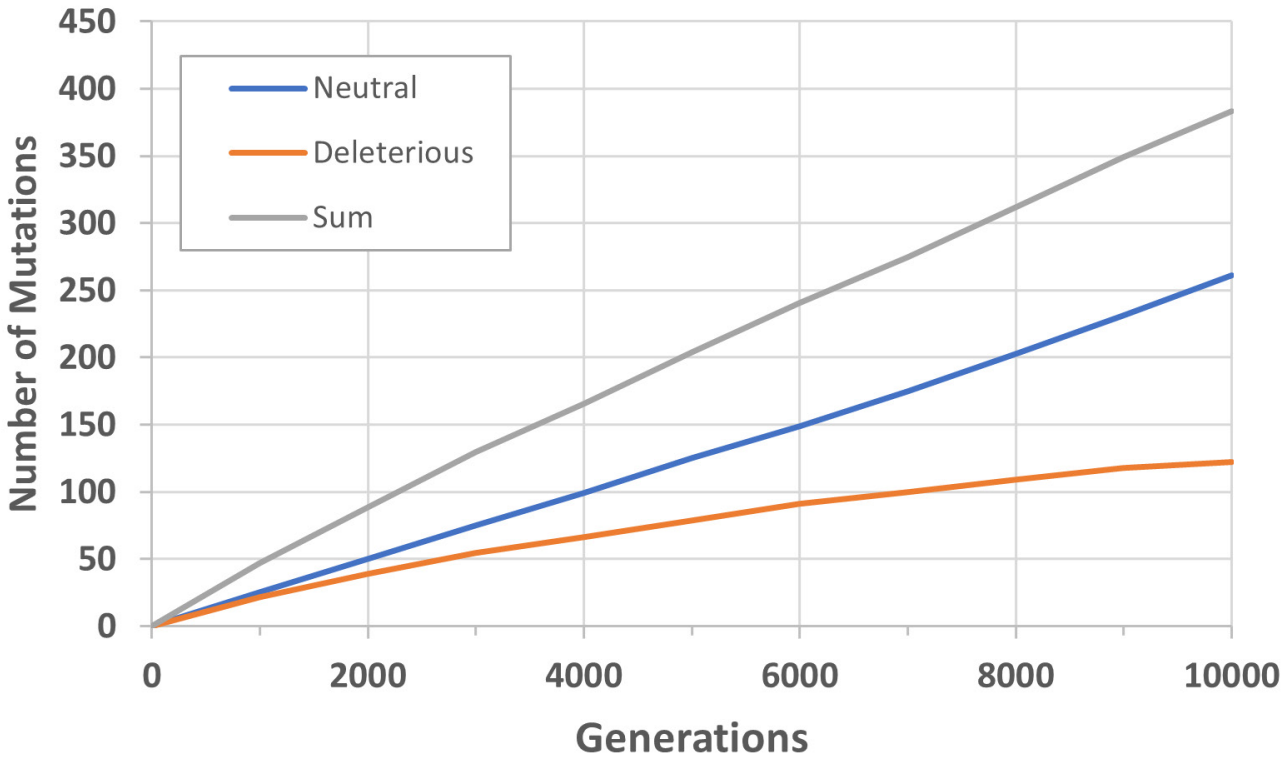

Figure 8. The accumulation of mutations in a Mendel model designed to assess mutation accumulation over an evolutionary timescale. These are the results of a single run with a mutation rate of 0.05 and a population size of 100,000. Data were plotted every 1,000 generations. Note that Mendel cannot model haploid systems, so all individuals would carry two versions of the mtDNA, thus complicating the analysis.

(Fig. 5). The loss of alleles due to genetic drift was highly consistent within each size class, increasing only slightly as populations approached the extinction threshold (Fig. 6). In the model behind Fig. 3 and Fig. 4, the rate of neutral alleles was set to 0.25. Without selection, the expected final ratio of neutral to deleterious alleles would be 0.25/0.75, or 1:3. The final ratio was 124,950:343,677, or 1:2.75. Selection was able to remove only about 8% of the deleterious alleles.

The average number of excess mutations in the runs where all mutations were neutral was 1,216 (+/- 558 SD). Given that individuals carried more neutral alleles than would be predicted by solely multiplying the number of generations by the neutral mutation rate, clearly one must account for the mutational half-life. A second population model (Carter 2019) was modified to track the lifespan of single mutations in human-like populations of various sizes (Fig. 7). N ranged from 10 to 100,000 and each model was run 1,000 times. There was a barely noticeable trend toward longer maximum and average lifetimes for neutral alleles (measured in generations) among the larger populations. Most new mutations were lost to drift within five generations. It took a little less time for this to happen in the smallest populations. This accounts for the slightly higher-than-expected mutation burden seen in the *Mendel* results. It takes a few generations to lose new mutations, so the total mutation count stands slightly above the long-term accumulation rate.

When modeling mitochondrial DNA over evolutionary timescales in *Mendel*, a mutation rate of 0.05 created a fixation rate of approximately 1 mutation every 2,700 years for n = 1,000, 5,000, and 10,000. This is between the phylogenetic rates published by Rieux et al. (2014) and Soares et al. (2009). However, the rate was 40% lower in the smallest population (n = 500) and there was zero substitution in the largest population (n = 100,000). Figure 8 shows a clear separation between the accumulation of deleterious and neutral mutations in the largest population. It also shows a gradual leveling off in the deleterious mutation curve, similar to the predictions of Soares et al. (2009). Some of this leveling off would have been due to the fact that all individuals started with a perfect fitness score. As mutations built up and fitness declined, selection would have increased. Thus, in these models there is a 'burn-in' time before the effects of selection can truly be measured. Yet, as stated above, no fixation was occurring in this population, even after approximately 300,000 years of model time. The smaller populations had a deleterious-to-neutral fixation ratio ranging from 0.4 to 0.9. Thus, even in the best-case scenario, at most 60% of the deleterious mutations can be removed.

Mendel is not actually set up to do the mitochondrial experiments described here. In its current configuration, there is no way to model asexual compartments. Thus, the individuals in these models carried *two* mitochondrial types and selection against the worst mitochondrial mutations would be mitigated by the second version carried by the individual. However, one might consider this a reasonable compromise after accounting for known interactions from the nuclear DNA, epigenetic effects, and environmental variability. Also, since there are upwards of 1,000 mitochondria per human cell, mitochondrial mutations start out in a hemizygous state by default. It takes several generations for any new mutation to go to fixation within an individ-

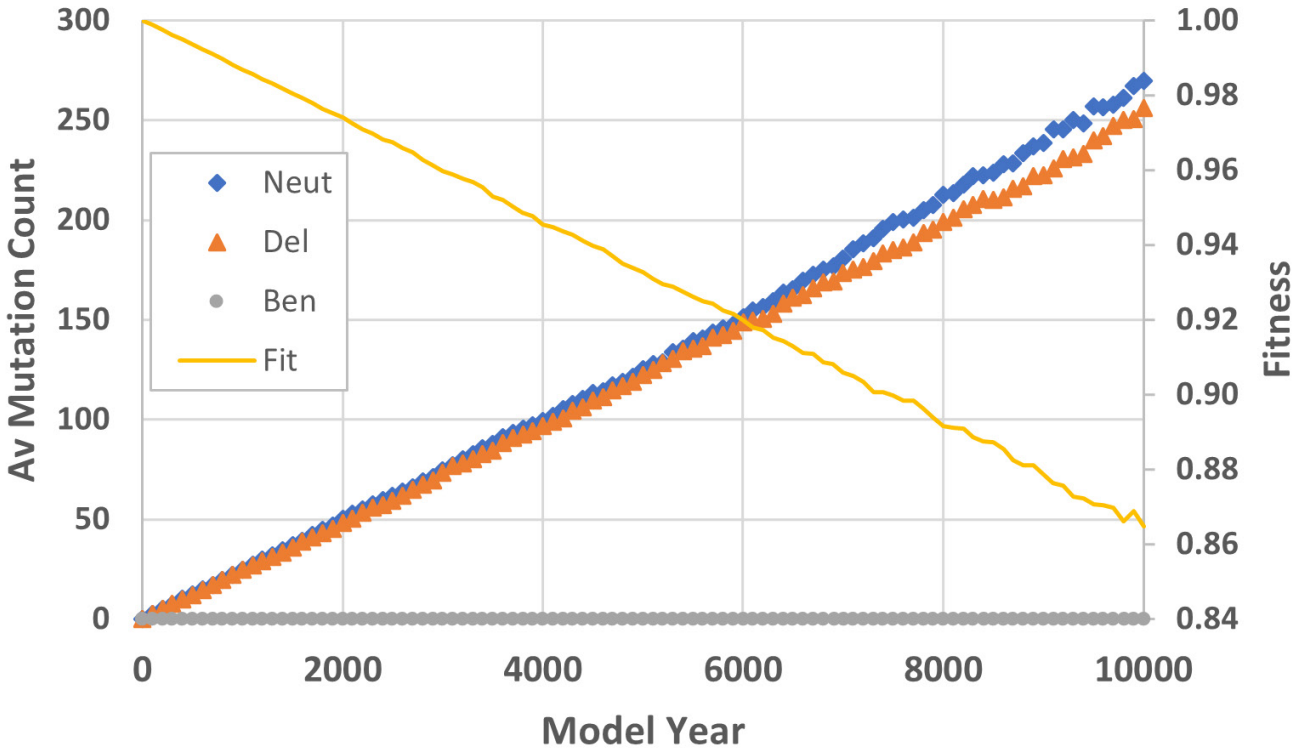

Figure 9: The accumulation of mutations in a haploid mitochondrial model. As above, these are the results of a single model run, but with a mutation rate of 0.005 and a population size of 10,000. Data were plotted every 100 generations.

ual's lineage. This is accelerated by a bottleneck every generation, where the number of mitochondria is reduced to approximately 100 during oogenesis (Li et al. 2018). Still, it takes several generations for a mutant lineage to become 'fixed' within a family line, meaning mitochondria are often found in a diploid state on the level of the individual.

Given this discrepancy, the population model of Carter (2019b) was further modified to include a similar style of probability selection as performed in *Mendel*. It was not trivial to achieve a population model that showed a significant reduction in deleterious mutations without causing extinction, and this is only amplified by the lack of recombination within the mitochondrial genome. The most effective method was to reduce the mutation rate while increasing the average mutation effect, but this quickly became non-biological, e.g., the mutation rate had to be set below 0.005. This is about two orders of magnitude below genealogical estimates (Fig. 9).

IV. DISCUSSION

Nearly all mutations, by evolutionary necessity, must be selectively neutral. Yet nearly all mutations are also expected to be deleterious (Sanford 2014). This is only becoming more obvious as additional functions are found for multiple genomic elements (Carter 2022b). Here, it was shown that selection can only remove a small number of the mutations that are bound to occur in any genome. This is not surprising, given what is already known (Carter 2019a).

And yet, the sheer number of new mutations in a population means that some mutations will be retained. In fact, a reasonable estimate of the mutation load of any given individual is obtained by simply multiplying the mutation rate by the number of generations that have elapsed. Thus, the formulas of standard population genetics are directly applicable to the question of the timing of Y Chromosome Adam and Mitochondrial Eve, with the caveat that selection will remove some small percentage of mutations. Genetic drift is almost irrelevant when considering the mutation load of any given individual. Thus, due to selection, the long-term, phylogenetic mutation rate will be slightly less than the short-term, genealogical mutation rate.

The degree of separation between these two rates depends on many factors, only some of which were modeled here. Yet, there is only so much that selection can do. Reproductive output is limited, and most mutations are expected to be lower than the selection threshold anyway.

Thus, the question comes down to 1) the real mutation rate and 2) the relative proportions of selectively neutral vs. non-neutral mutations. Yes, selection can remove a certain proportion of deleterious mutations, but if the rate at which these occur is relatively low, there is nothing for selection to act upon. To see a significant difference between the genealogical mutation rate and the phylogenetic mutation rate, the proportion of deleterious alleles would need to be much higher than theory allows. Selection is also more efficient in smaller populations, but so is fixation, which increases the risk of extinction. Yet, even in the smallest populations with high rates of deleterious alleles, the ratio of observed to expected fixed deleterious alleles was greater than 0.9. There is no way to remove a higher proportion of deleterious mutations, and those populations all went extinct!

Several anti-creationists have made the mistake of assuming that, since most mutations are lost, the phylogenetic mutation accumulation rate is necessarily much slower than the genealogical mutation rate. This exemplifies a gross misunderstanding of the mathematics

of population genetics. In fact, the rate of change within a species (the fixation rate) has little to do with the rate of change within the individuals inside the population (the mutation rate). The fixation rate is extremely slow in large populations, but individuals are always accumulating mutations. An individual carries the new mutations they were born with plus whatever mutations they inherited from their ancestry. Because of this, the timing of the Y chromosome and mtDNA common ancestors are independent of the fixation rate; they only depend on the mutation rate.

There is a fine balance between mutation, selection, and long-term survival. Generally, selection is invoked as a means of both advancing a species (e.g., positive selection) and protecting a species against decay (e.g., purifying selection). Yet, selection is limited in its power (due to epistasis, epigenetics, and other sources of 'noise') and speed (due to limits of reproductive output, see ReMine 2005). Genetic drift also fails to account for the discordance between the genealogical and phylogenetic mutation rates. The evolutionary community is not unaware of these difficulties, yet they persist in their belief that, given enough time, the genealogical and phylogenetic mutation rates will diverge significantly. In computer models, there are ways to maximize the difference (e.g., by lowering the base mutation rate and increasing the negative effects of deleterious mutations), but the question of biological reality always looms over the results. The constraints of biology severely limit evolutionary models, to the point where basic mathematics argues strongly against all long-term evolutionary ideas.

The problem is amplified for haploid compartments like mitochondria and Y chromosomes. Recombination has a real, measurable, long-term benefit in helping to remove deleterious alleles. Haploid systems do not undergo recombination. Thus, mutations accumulate in a ratchet-like way (Rupe and Sanford 2013) and all lineages will be picking up deleterious mutations over time. The net effect is a downward trend. Worse, the negative effects of mutations cannot be masked by alternate alleles, as in haploid systems. Due to these factors, attempts to model the separation of neutral and deleterious alleles over time (e.g., Fig. 9), were hampered. Many model settings resulted in population extinction before the proscribed 10,000 generations (300,000 years) was reached, and this was after the mutation rate was reduced to 0.005 (one new mutation in every 200 births, with half of being perfectly neutral) or less. Note the declining fitness trend line in Fig. 9 and the fact that the x-axis is in years, not generations. The population in this model did not make it to 300,000 years. It was possible to reduce the average effect of deleterious mutations, but this would lead to even less removal of these mutations over time. It was also possible to reduce the mutation rate, but that would diverge even further from biological reality. In the end, causing a divergence between the genealogical and phylogenetic mutation rates is a non-trivial matter. There is no reason to suspect the two would be significantly different over long timespans. Hence, the genealogical rate stands as a valid method of computing ancestral events and both Mitochondrial Eve and Y Chromosome Adam must have lived in the recent past.

Several skeptics have attempted to argue that there is no 'perfect' fitness, as evolution is considered to be a continual process of mutation and selection over millions of generations. Thus, they claim, it is incorrect to start all individuals with no deleterious or beneficial alleles. However, in these models, 'fitness' is arbitrary. It is simply a measure of the relative reproductive potential of any individual with respect to the other individuals alive at the time. Yes, tracking changes in fitness allows us to see long-term trends, but reproductive potential is always in terms of the contemporaneous population. Also, given long run times, sufficient mutational 'burn in' occurs, such that the population contains a range of fitness scores, similar to what is assumed in evolutionary models.

Note also that lethal mutations were not taken into account in this study. By default, any mutation that causes death, that prevents pregnancy, or that causes severe malformations or intellectual disability, will be filtered out of the population instantaneously. These mutations reduce reproductive output but are never subject to selection in the way that it is modeled here (e.g., via an annual risk of death).

The mutation rate per generation is approximately 60 in the nuclear genome, 1 in the Y chromosome, and 0.5 in the mitochondrial genome. Every one of these estimates puts Adam and Eve within a biblical time frame, even if we were to reduce the mutation rates by an order of magnitude. Natural selection cannot remove most of these mutations, so the burden of proof is on the evolutionary community to explain the discordance between the genealogical and phylogenetic mutation rates.

V. CONCLUSIONS

In conclusion, the short-term, measurable, genealogical mutation rate is a serious challenge to evolutionary history. The long-term mutation accumulation rate should equal the base mutation rate less the proportion of deleterious alleles that can be removed by selection. Yet, even if selection were 100% efficient at removing all deleterious alleles, it would have no effect on neutral alleles. Given that most alleles are selectively neutral, only a small proportion of all mutations can be removed. It would take very little time to accumulate the number of differences seen in extant Y and mitochondrial chromosomes. The amount of diversity seen in human autosomes could also be explained in a biblical timeline. When one considers that much of that diversity was probably created by God and placed directly into Adam and/or Eve, it would be trivial to explain what remains.

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THE AUTHOR

Robert Carter received his PhD from the University of Miami in 2003. With a background in genetics, gene cloning and sequencing, and transgenics, he soon began to work on the GENE project team (ICR), then as a speaker for Creation Ministries International. Over the years, he has published multiple papers directed toward building a biblical model of human genetic history.