Human Genetics and Origins

We are told that we are 99% chimpanzee. We are also told that we are up to 4% Neanderthal. How can those statements both be true? Aren't we more closely related to Neanderthals than to chimpanzees? Why the disconnect? This suggests that perhaps the standard narrative needs examination.

First, an article in *Science* states that the 99% myth is just that, a myth.[1] The article details how in 1975 in S*cience* Allan Wilson and Mary-Claire King wrote an article claiming that there was a 1% difference between the genomes of chimpanzees and humans. Actually, to be precise, they showed an approximately 1% difference between the coding regions of selected genes.[2] Thus the myth was born. Quoting Cohen,
"At the time, that was heretical," says King, … Subsequent studies bore their conclusions out and today we take as a given that the two species are genetically 99% the same.

Again according to Cohen, it was once a useful myth:
"For many years, the one percent difference served us well because it was underappreciated how similar we were, "says Pascal Gagneux, a zoologist at UC San Diego. "Now it's totally clear that it's more a hindrance for understanding than a help."

Again quoting Cohen,
"But truth be told, Wilson and King also noted that the one percent difference wasn't the whole story. They predicted that there must be profound differences outside genes – they focused on gene regulation – to account for the anatomical and behavioral disparities between our knuckle-dragging cousins and us".

And further studies have suggested that 1% is a serious underestimate of the differences. According to the Chimpanzee Sequencing and Analysis Consortium,[3] while there are 1.23% single nucleotide polymorphisms (SNPs, differences where there is more than one nucleotide

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[1] Cohen J, 2007. Relative differences: the myth of 1%. *Science* 316:1836. Available at https://www.science.org/doi/10.1126/science.316.5833.1836

[2] King M-C, Wilson AC, 1975. Evolution at two levels in humans and chimpanzees: Their macromolecules are so alike that regulatory mutations may account for their biological differences. Science 188:107-16. Available at https://www.science.org/doi/pdf/10.1126/science.1090005?casa_token=CnQwt4iWDw4AAAAA:0Mm3YTCgmT8O8wOb1t0iddwC5aFNeArKCEYHctvXXizQ1zgPy5cBxGkTucyDTmxNiMgmJ70fWwf-vJlG

[3] The Chimpanzee Sequencing and Analysis Consortium, 2005. Initial sequence of the chimpanzee genome and comparison with the human genome. *Nature* 437, 69–87. Available at https://www.nature.com/articles/nature04072#citeas

in a position, in this case chimpanzees have one nucleotide and humans have another), probably reducible to 1.06% because in some of those cases there are SNPs within humans or within chimpanzees, there are also 1.5% indels (where code has either been inserted in one species or deleted in the other) in each species, for 3% total, making a total difference of 4%, meaning that we are only 96% identical.

But this is true only of the genes we share. Cohen points out that according to another study,[4] "Our results indicate that the human genome contains 1,418 genes—6.4% of all genes—that do not have orthologs in the chimpanzee genome (689 gains in humans+729 losses in chimpanzee/22,000 total genes)." That means that for these coding genes, there is zero correlation. So if one is being fair about the coding genes, one must multiply 93.6% by 95.96%, which gives us 89.9% similarity in the coding genes, and we are less than 90% chimpanzee in our coding genes.

In fact, now that virtually the entire genetic code of both chimpanzees and humans is available, two creationist scientists[5] have used a computer program to find parallels between the two whole genomes, and come up with an identity of 84%. That there are parallels is beyond question, but that they approach 99% is demonstrably inaccurate.

So why does the myth persist? Perhaps because it can be used as a potent argument. At least in this case, the current scientific consensus is not to be blindly trusted.

We are told that the population of hominids never dropped below 10,000, and that there never was a first man or a first woman, from whom everyone has descended, and that the story of Adam and Eve is a myth. Well, maybe not so much. The current story is that in our mitochondria, inherited for practical purposes solely from our mothers, we do go back to one woman; all other female lines eventually died out, and although other genes could survive in males, the mother-daughter-granddaughter etc. chain eventually disappeared for all but one line,[6] with the original female progenitor being dubbed Mitochondrial Eve.

According to the article, this woman lived in Africa around 200,000 years ago. To quote Cann *et al.*, "Assuming a rate of 2%-4% per million years, this implies that the common

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[4] Demuth JP *et al.,* 2006. The Evolution of Mammalian Gene Families. *PLoS ONE* 1(1): e85. Available at https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0000085

[5] Buggs R, online: https://richardbuggs.com/2018/07/14/how-similar-are-human-and-chimpanzee-genomes/ ; Tomkins JP, 2018. Comparison of 18,000 De Novo Assembled Chimpanzee Contigs to the Human Genome Yields Average BLASTN Alignment Identities of 84%. *Answers Research J* 11:205-209. Available at https://answersresearchjournal.org/comparison-chimp-contigs-human-genome/

[6] Cann RL *et al.*, 1987. Mitochondrial DNA and Human Evolution. *Nature* 325:31-36. Available at https://www.nature.com/articles/325031a0.pdf

ancestor of all surviving mtDNA types existed 140,000-290,000 years ago." This was not what standard theory was expecting. According to standard theory, there were humans scattered around the old world for perhaps a million years, and some of them should have left direct female lines all the way to the present. The idea that within perhaps 100,000 years all these female lines should have just disappeared seems highly improbable. Again according to Cann *et al.*,

An alternative view of human evolution rests on evidence that Homo has been present in Asia as well as in Africa for at least one million years[48] and holds that the transformation of archaic to anatomically modern humans occurred in parallel in different parts of the Old World[33,49]. This hypothesis leads us to expect genetic differences of great antiquity within widely separated parts of the modern pool of mtDNAs. It is hard to reconcile the mtDNA results with this hypothesis.

We'll come back to the Africa part. For now, it is sufficient to note that the mutational rate for mitochondria was originally calibrated by the standard geological time scale. When calibrated to actual history, it turns out to be much higher than expected.[7] This gives rise to several expressions of surprise in the literature. To quote Parsons *et al.*,

"… Taken together, our data indicate a remarkably high substitution rate, ~one in 33 generations. Assuming a generation time of 20 years, this extrapolates to a substitution rate of 2.5/site/Myr (95% confidence interval, 1.2-4.0/site/Myr). …

Skipping down a few paragraphs, we find,

"The observed substitution rate reported here is very high compared to rates inferred from evolutionary studies. … 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 ([most recent common ancestor] of only ~6,500 y.a., clearly incompatible with the known age of modern humans.

While our results are at odds with those of phylogenetic studies, they are in excellent agreement with a recent report that also directly measured the CR substitution rate[37]. …".

They then ask the question, "What could account for the disparity between the observed substitution rate and those derived from phylogenetic analyses?" They suggest the possibility of "mutational 'hot spots'"

This conundrum led Ann Gibbons to comment,[8]

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[7] Parsons TJ *et al.*, 1997. A high observed substitution rate in the human mitochondrial DNA control region. Nature Genetics 15:363-368. Available at https://www.religiousforums.com/data/attachment-files/2020/02/42408_3fb671ab1438a4dc912e38c148f7eca1.pdf

[8] Gibbons A, 1998. Calibrating the Mitochondrial Clock. *Science* 279: 28-29. Available at https://pdfs.semanticscholar.org/6984/231c83877906a2a272696edc21ada3382f61.pdf

"Regardless of the cause, evolutionists are most concerned about the effect of a faster mutation rate. For example, researchers have calculated that "mitochondrial Eve"—the woman whose mtDNA was ancestral to that in all living people—lived 100,000 to 200,000 years ago in Africa. Using the new clock, she would be a mere 6000 years old.
No one thinks that's the case, but at what point should models switch from one mtDNA time zone to the other"?

Well, almost no one thinks that's the case. Maybe the empirical time zone is the real one.

It turns out that this rapid rate of mitochondrial mutation is not true just for humans.[9]
"… Analyses of the mitochondrial control region, for example, have yielded mutation rates as high as 32%–260% per million years in humans (Parsons et al. 1997; Sigurdardóttir et al. 2000; Howell et al. 2003) and 95% per million years in Adélie penguins (Lambert et al. 2002). These estimates (of the mutation rate in the control region) vastly exceed the traditionally recognized substitution rate of 1% per million years for protein-coding mitochondrial DNA
It turns out that this problem of a recent Mitochondrial Eve is there not only for humans, but for over 90% of all species. In 2018 we discovered that the vast majority of species appeared to be 100,000 to 200,000 years old by mitochondrial barcode.[10] The mitochondrial barcode, a section of the mitochondrial genome coding for part of cyclooxygenase, has proved useful for distinguishing species, but somewhat surprisingly, the intraspecies variation is small.[11] As Agence France-Presse quoted David Thaler, "This conclusion is very surprising, and I fought against it as hard as I could," But if the mitochondria of other species have faster mutation rates than expected, the most recent common ancestor of 90% of species could also date to around 6,000 years. This is not what the standard model predicted (reference)".

The Y chromosome, passed down from father to son, gives a similar estimate.[12] Underhill's 95% probability interval was 40,000-140,000 years. Other estimates[13] differ somewhat, but all agree that Y chromosome divergence happened either simultaneously to or after

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[9] Ho SYW *et al.*, 2005. Time Dependency of Molecular Rate Estimates and Systematic Overestimation of Recent Divergence Times. *Molecular Biology and Evolution* 22:1561–1568. Available at https://academic.oup.com/mbe/article/22/7/1561/974191?login=false

[10] Stoeckle MY, Thaler DS. Why should mitochondria define species? *Human Evolution* 33:1-30. Available at https://www.biorxiv.org/content/biorxiv/early/2018/03/07/276717.full.pdf

[11] For a more popular explanation, see https://m.phys.org/news/2018-05-gene-survey-reveals-facets-evolution.html

[12] Underhill PA *et al.*, 2000. Y chromosome sequence variation and the history of human populations. *Nature Genetics* 26:358–361. Available at ResearchGate

[13] For example, Posnik GD *et al.*, 2013. Sequencing Y chromosomes resolves discrepancy in time to common ancestor of males versus females. Science 341:562-5. Available at https://www.science.org/doi/full/10.1126/science.1237619?casa_token=zNFAzWbE2jEAAAAA:H0je7LkYCqdQDYiQmt_4JA-c91iIb7jSTchfa1wwam1oJ5M7g2Nvh32-JgfVb84Aml9OP6U7rRn6aiQz

mitochondrial divergence. From a creationist perspective, a slight difference would not be surprising, as Y Chromosome Adam would actually be Noah, who fathered all the males on the Ark, whereas the three sons' wives could possibly be divergent back to Eve.

Jeanson and Holland argue for a more rapid molecular clock than the standard one.[14] Briefly, their argument is that there were 4 studies of mutation rates in the human Y chromosome, by Xue *et al.*,[15] Karmin *et al.*,[16] Helgason *et al.*,[17] and Maretty *et al.*[18] What Jeanson and Holland considered the low coverage studies (those whose duplicating of portions of the Y chromosomes was less than 16, making it easier to miss mutations), Xue *et al.* and Helgason *et al.*, gave mutation rates of around 3×10^{-8}, whereas the higher coverage studies (>35), Karmin *et al.* and Maretty *et al.*, gave 3×10^{-7} and 5×10^{-7}, respectively. If one trusts the higher quality studies, the age of Y Chromosome Adam lowers to around creationist expectations. Further data may help to refine the experimental mutation rate.

Figure 1. From Underhill et al., see note 12 Figure 2. From Cann et al., see note 6

It has been observed that for both the Y chromosome and the mitochondrion human trees have consistently displayed a wider divergence in sequence in African lineages than in European or Asian lineages, or those from Asia, the Pacific islands (including Australia), or the New World. The standard interpretation is that the lineages came out of Africa, leaving deeper branches there. A short-age creationist interpretation would seem to predict that the

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[14] Jeanson NT, Holland AD, 2019. Evidence for a human Y chromosome molecular clock pedigree based mutation rates suggest a 4,500-year history for human paternal inheritance. *Answers Res J* 12:393-404. Available at https://ebcky.com/2019/12/04/evidence-for-a-human-y-chromosome-molecular-clock-pedigree-based-mutation-rates-suggest-a-4500-year-history-for-human-paternal-inheritance/

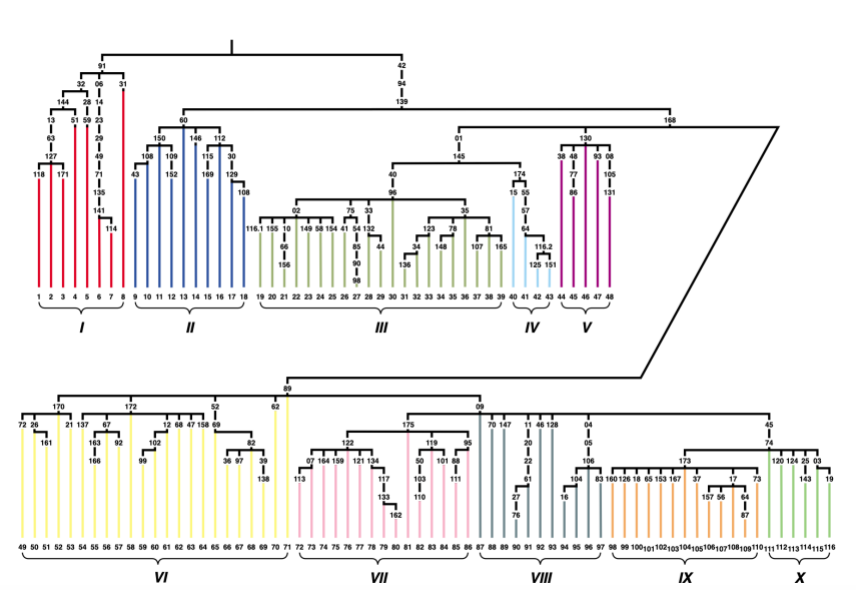
[15] Xue Y *et al.*, 2009. "Human Y Chromosome Base-Substitution Mutation Rate Measured By Direct Sequencing In a Deep-Rooting Pedigree." *Curr Biol* 19:1453–7. Available at https://www.sciencedirect.com/science/article/pii/S0960982209014547

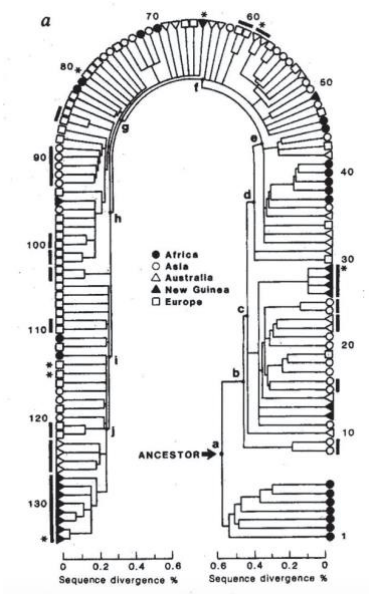
[16] Karmin M *et al.*,2015. A recent bottleneck of Y chromosome diversity coincides with a global change in culture. *Genome Res* 25:459-66. Available at https://genome.cshlp.org/content/25/4/459.full.pdf

[17] Helgason A *et al.*, 2015. The Y-chromosome point mutation rate in humans. *Nature Gen* 47, 453-7. Available at https://www.nature.com/articles/ng.3171

[18] Maretty L *et al.*, 2017. Sequencing and De Novo Assembly of 150 Genomes from Denmark as a Population Reference. *Nature* 548:87-91. Available at https://www.nature.com/articles/nature23264

mutation rate in Africa was faster than that in the rest of





the world, perhaps because of hereditary factors, and/or perhaps because of environmental factors, and this prediction could be tested.

One of the surprises with the Y chromosome is that its structure is remarkably divergent between chimpanzees and humans.[19] In fact, in a news report accompanying the article,[20] David Page, the final author of the main article, is quoted as saying that "The common chimp (*Pan troglodytes*) and human Y chromosomes are 'horrendously different from each other'. … 'It looks like there's been a dramatic renovation or reinvention of the Y chromosome in the chimpanzee and human lineages.'"

To continue the quote,
"Even the portions that do line up have undergone erratic relocation. In the only other chromosome to have been sequenced to the same degree of completeness in both species, chromosome 21, the authors found much less rearrangement.
"If you're marching along the human chromosome 21, you might as well be marching along the chimp chromosome 21. It's like an unbroken piece of glass," says Page. "But the relationship between the human and chimp Y chromosomes has been blown to pieces."

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[19] Hughes JF et al., 2010. Chimpanzee and human Y chromosomes are remarkably divergent in structure and gene content. Nature 463(7280): 536-539. Available at http://www.nature.com/news/2010/100113/full/463149a.html

[20] The fickle Y chromosome: Chimp genome reveals rapid rate of change. Available at http://www.nature.com/news/2010/100113/full/463149a.html

The article goes on to observe,

"Surprisingly, however, > 30% of chimpanzee MSY [male-specific Y; the vast majority of the Y chromosome that does not cross over with the X chromosome] sequence has no homologous, alignable counterpart in the human MSY, and vice versa (Supplementary Fig. 8 and Supplementary Note 3). In this respect the MSY differs radically from the remainder of the genome, where < 2% of chimpanzee euchromatic sequence lacks an homologous, alignable counterpart in humans, and vice versa[15].

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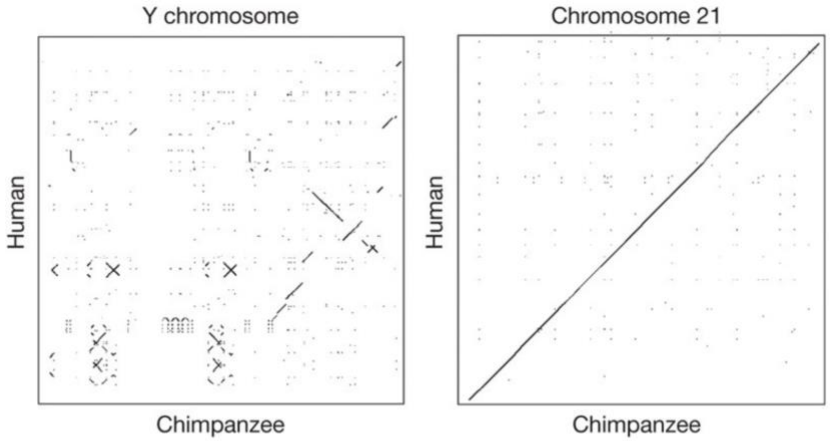
"… Indeed, at six million years of separation, the difference in MSY gene content in chimpanzee and human is more comparable to the difference in autosomal [non-sex chromosome] gene content in chicken and human, at 310 million years of separation[26].

This sounds wildly exaggerated until you see the accompanying graphs:

Figure 3. From Hughes *et al.*, see note 19



Each dot represents 100% chimpanzee–human identity within a 200-base-pair (bp) window. In the Y-chromosome plot, the human chromosome is oriented with short arm to top and long arm to bottom, and the chimpanzee chromosome is oriented with short arm to left and long arm to right. For chromosome 21, which is acrocentric, the plot represents only the long arm.

Note that Chromosome 21 is close to identical in both sequence and structure. But note also that the Y chromosome is completely disorganized. In fact, the expected orientation in the Y

chromosome is a match from upper left to lower right, and it looks like what little organization is left is mostly organized from upper right to lower left. Why the orientation in the two chromosomes is not the same in Figure 3 is not clear. Since the Y chromosome contains roughly 1/50th of the genome, it can take care of close to a 1% difference between the human and chimpanzee genomes all by itself.

Finally, it has been discovered that at 481 sites, human, rat, and mouse DNA is identical for over 200 bases at each site.[21] To quote the abstract of Snetkova *et al.*,
Indent
Across the human genome, there are nearly 500 'ultraconserved 'elements: regions of at least 200 contiguous nucleotides that are perfectly conserved in both the mouse and rat genomes. Remarkably, the majority of these sequences are non-coding, and many can function as enhancers that activate tissue-specific gene expression during embryonic development. From their first description more than 15 years ago, their extreme conservation has both fascinated and perplexed researchers in genomics and evolutionary biology. The intrigue around ultraconserved elements only grew with the observation that they are dispensable for viability. Here, we review recent progress towards understanding the general importance and the specific functions of ultraconserved sequences in mammalian development and human disease and discuss possible explanations for their extreme conservation.

The requirement for 200 bases is completely arbitrary. As Snetkova *et al.* say,
"… For example, lowering the length threshold for human–mouse–rat comparisons to 100 bp increased the number of identified elements by an order of magnitude, to more than 5,000 (ref.[10]).

It isn't like the areas cannot mutate:
"… In other words, common variants are particularly depleted in ultraconserved loci, whereas extremely rare variants occur at levels close to those observed elsewhere in the genome, excluding the possibility that ultraconserved elements are generally protected from mutation. …"

Nor is it that mutating these areas kills the organism:
"… We highlight their importance in establishing normal development while having no obvious impact on viability, and we investigate possible explanations for their perfect sequence conservation. …"

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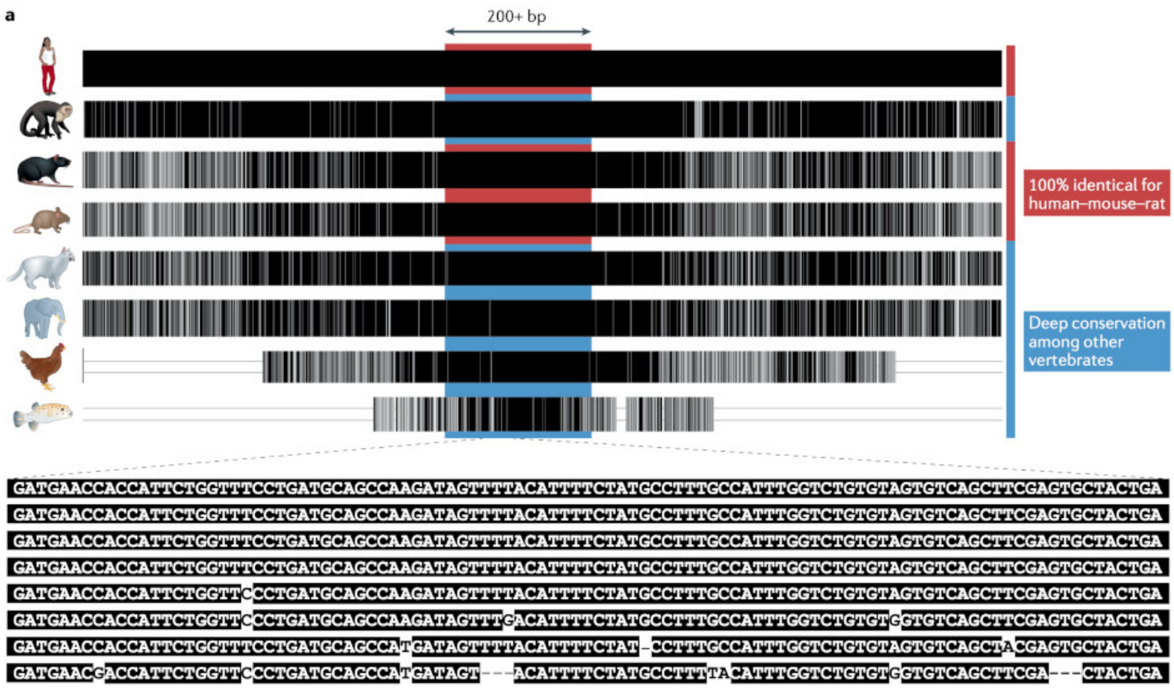
[21] Snetkova V, Pennacchio LA, Visel A, Dickel DE 2022. Perfect and imperfect views of ultraconserved sequences. *Nat Rev Genet* 23(3):182-194. Abstract available at https://www.nature.com/articles/s41576-021-00424-x

Figure 4. From Snetkova *et al.*, see note 21



In fact, one can completely cut these segments out:
"… Remarkably, mice that were homozygous-null for individual ultraconserved enhancers (or hemizygous-null males for enhancers on the X chromosome) showed no indication of increased prenatal lethality, were fully viable at birth and lived well into adulthood. Assessments of pathology, growth and gene expression did not reveal obvious detrimental phenotypes resulting from the loss of these enhancers. Given the extreme sequence conservation of these loci, the lack of apparent phenotypes was quite surprising to many in the field and led to various hypotheses as to the reason for this observation. …"

In addition, it's not just removing them that is harmless; one can mutate them as well
"… Surprisingly, even at a substantial mutation rate of 5%, nearly half of the enhancers (44%) remained active. In one extreme case, an ultraconserved enhancer showed residual tissue-specific activity upon mutation of 20% of ultraconserved base pairs. These results indicated that ultraconserved enhancers do not commonly lose their enhancer function upon even significant levels of mutation. …

To summarize,

"Currently, unanswered questions remain about why ultraconserved sequences are so highly conserved and exactly what function evolutionary selection is acting on to maintain this conservation. To date, half of all ultraconserved enhancers deleted in mice have not been shown to result in a potentially detrimental phenotype, and there has been no direct demonstration that loss of any ultraconserved enhancer results in reduced viability, fertility or fecundity. …"

A little thought will reveal why the constant expressions of surprise. Standard theory is that the line that would eventually give rise to humans diverged from the line that eventually gave rise to rats and mice around 100 million years ago, with rats and mice diverging some 30 million years ago. Presumably, this divergence was in a small, shrew-like animal. Shrews have 1 to 3 generations per year; rats and mice can have up to 5 generations per year, although that is probably an overestimate, as grandpa and grandma mouse are still able to have babies. As a conservative estimate, let's say that there is one generation per year. There are about 100 mutations per generation in the entire genome (2.5 million bases in the mouse, 2.75 million bases in the rat). In 100 million years, just in the rat line, we would expect 100 mutations per generation times 100 million generation, or 10 billion mutations. That works out to 3.6 mutations per base. It is hard to see how there would be 10 bases untouched, let alone 200. The only way one could keep a particular 200 base pair stretch of DNA intact for 100 million years is if one killed all the rats (and mice and humans) with any mutations in these areas, or made them infertile, or at least gave them a measurable disadvantage in reproduction. Yet that does not appear to be the case.

On the other hand, consider a Creator who made rats, mice, and humans separately some 6,000 years ago, or even 10,000 years ago, using the same base pairs in a given area of the genome in all three species. Say there were 5 generations per year, and 100 mutations per generation, That works out to be 100 x 5 x 10,000, or 5 million mutations, or one in every 550 bases. One could easily miss a particular 200 bases in all three species by random chance. It would seem to be much easier to explain identical base pairs in all three species with a short-age hypothesis than with a long-age one.

In summary, you are not being told the whole story. We are not 99% chimp; it is closer to 80-90%. Even by evolutionary standards, Mitochondrial Eve and Y Chromosome Adam are not as old as expected. If empirically determined mutation rates are correct, they date in the 6,000 year range. For mitochondria, this is true for over 90% of all species. The difference between human and chimpanzee Y chromosomes is "horrendous" and suggests the possibility that the standard story of chimp and human Y chromosome evolution is grossly inaccurate. And multiple large segments of identical DNA in humans, rats, and mice suggest a much shorter time frame than the conventional one. There is still research to be done, for example on differential mutation rates. But mostly unpublicized studies in human genetics suggest that a short-age creationist position may be more defensible than the standard one