Genetic variability and human history

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Observations concerning genetic variability and linkage disequilibrium in the human genome point to a relatively recent population bottleneck in humankind, consistent with the Flood. A number of genetic processes exist which could have produced the necessary variation observed since the Flood, including transposable elements and environmentally induced mutations and recombinations. Finally, the observation that Africans are more diverse than all other human populations need not imply a longer population history, and is consistent with the splitting of human groups at Babel.

The population bottleneck at the Flood

As more has been learned about the human genome, evidence has mounted which suggests a relatively recent population bottleneck in human history—that is, a severe reduction in population size followed by an expansion. This is precisely what would be expected if the Flood had occurred. Low levels of diversity in humans have indicated the likelihood of such an event. For example, Knight *et al*. studied over 55 kb (kilobases, 1,000 base pairs) from diverse human populations and found that genetic diversity was exceedingly low, most individuals being identical. They commented that

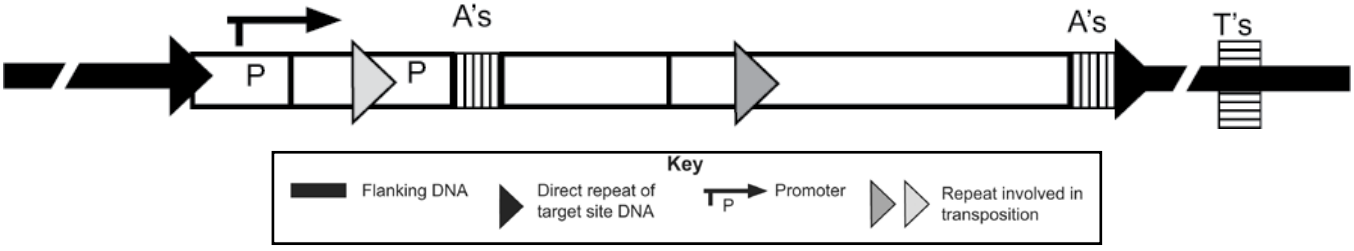
> 'if human races evolved in widely separated geographic regions for over 1.5 Myr, *Alu* sequences* would reveal substantial interallelic divergence and overall nucleotide diversity. On the other hand, if the human autosomal* genetic complement has undergone a relatively recent worldwide replacement, we should expect only very low levels of differences among populations and individuals.'[1]

This is, of course, exactly what they observed and precisely what biblical history would predict. Evolution, on the other hand, did not predict this. There is also less variation in mtDNA (mitochonderal DNA) than was expected.[2] Additionally, non-coding DNA is very similar, running counter to what evolution expected: 'vertebrate genomes contain thousands of noncoding sequences that have persisted virtually unaltered for many millions of years. These sequences are much more highly conserved than those coding for proteins, which was *totally unexpected*' [emphasis mine].[3]

Other evidence of genetic similarity has been discussed in the context of human evolution.[4,5] Ambrose states that 'Low genetic diversity in human populations is thus largely attributable to the recent origins of modern humans.'[6] This evidence is often attributed specifically to the eruption of Mount Toba in Sumatra ~70,000 years ago. This evidence will instead be discussed in the context of the Flood.

Evidence concerning mtDNA also supports ideas of a population bottleneck. Many sites have been labelled mutational hot spots—locations in the genome at which mutations occur more quickly than average. It has been suspected, however, 'that the so-called hypervariable sites in mtDNA might not be mutation hotspots, but rather ancient mutations that might have become distributed among unrelated haplogroups* in living peoples'.[7] The means of distribution could easily have been the Flood and/or the Babel incident, when populations split and bottlenecks occurred. Parsons *et al.* also commented that 'the "hot spot" hypothesis, in the absence of additional elements, does not seem a sufficient explanation for the high observed substitution rate'.[8]

Some of the most significant evidence supporting the population bottleneck comes from linkage disequilibrium in the nuclear genome. Genes are located on chromosomes in cells, and these genes may either be distant or close to each other. All of the genes that are located on one chromosome



Structure of a typical Alu *family sequence. This family consists of short, non-mobile DNA elements that occur almost a million times in the human genome. These sequences have been found to be highly conserved among populations and individuals, which indicates that the human genome has undergone a relatively recent worldwide replacement.*

* Items with an asterisk are defined in the glossary at the end of this article.

are said to be linked. When cells divide through meiosis, crossing over (or genetic recombination) often occurs in nuclear DNA. This involves two chromosomes aligning and swapping segments of DNA, resulting in genes getting 'shuffled around'. The closer that two genes are to one another, the higher the chances are that they will be inherited together—because they are close, it is unlikely that they will be separated during crossing over. When this holds true, the genes are said to be in linkage disequilibrium (or LD)—a state in which they are not thoroughly mixed, but tend to be inherited together in populations.[9] Over time, crossing over 'mixes up genomes so that they become more homogeneous'.[10]

Single nucleotide polymorphisms (hereafter SNPs, pronounced 'snips', which account for the bulk of diversity in humans) are point mutations that are relatively common to a genome, being present at a frequency of roughly 1% of the population.[11] The discussion of LD, above, also applies to SNPs. Many SNPs are associated with each other on chromosomes, and have been inherited together in populations. These 'blocks' of SNPs are called haplotypes*. LD refers to the existence of such non-homogeneous relationships. Crossing over breaks down ancestral haplotypes over time (separates the SNPs), and therefore decreases the extent of LD. Consider shuffling a deck of cards as an analogy. Imagine that the five of hearts is in very close proximity with the nine of spades. The closer these cards are together, the less likely it is that they will be separated by the shuffling of the deck (comparable to crossing over). Until all such relationships are broken between cards, they would not be thoroughly mixed, and this would be a state comparable to LD. In the same way, the distance that LD extends across the genome is broken down over time, and there is a decline in LD with increasing distance.

In 1999, Kruglyak[12] predicted that LD would extend about 3 kb unless the population studied had experience a 'very narrow bottleneck'. The bottleneck 'could be associated with the emergence of modern humans from Africa, in which case the pattern of linkage disequilibrium it left would be shared among many different human populations'.[13] If LD extending further than 3 kb would indicate a severe bottleneck, one has most certainly been uncovered in recent research. For example, Dunning *et al.*[14] published results which found that LD could extend even up to 500 kb, and that this evidence 'could reflect a more complex demographic history than that modeled by Kruglyak'.

Abecasis *et al*.[15] uncovered similar data when they found 'that significant disequilibrium in [three] regions can be detected at distances of 30–300 kb, which is an order of magnitude greater than those predicted by simulation' by Kruglyak. Similar conclusions have been reached by others.[16–18]

Reich *et al*.[19] also discovered that LD extends >100 kb in some cases in Europeans, and that blocks of LD are generally large in humans. In their words:

> 'Why does LD extend so far? LD around an allele* arises because of selection or population history—a small population size, genetic drift or population mixture—and decays owing to recombination, which breaks down ancestral haplotypes. The extent of LD decreases in proportion to the number of generations since the LD-generating event. The *simplest explanation* for the observed long-range LD *is that the population under study experienced an extreme founder effect or bottleneck:* a period when the population was so small that a few ancestral haplotypes gave rise to most of the haplotypes that exist today' [emphasis added].

Their simulations dated this bottleneck at 27,000–53,000 years before the present, and they added that the degree of LD is too high to have been caused by population mixture alone. However, there was much less LD present in Africans (see below).

Clearly, evidence supports the notion of a recent bottleneck in human history. Patterns of LD are quite irregular, however, and this could be due in part to differences in recombination rates over the genome. Arnheim *et al*.[20] found that 'Hot spots (and cold spots) of recombination have been observed in all experimental organisms that have been studied in any detail', and that 'quite dramatic hot spots (even recombination rates thousands of times greater than the genome average) might be completely missed in low-resolution analyses'. In fact, it is possible that recombination itself causes mutations.[21]

Although it is true that recombination hotspots could be a factor in the large extent of LD observed, it is not necessary. Because human populations share much of their biological history (even within an evolutionary framework), 'even large differences in the recombination landscape of extant populations may be difficult to detect from LD data'.[22] LD can be inflated by either inbreeding, population structure or bottlenecks. Clearly, high variation in recombination rates in the genome is just one interpretation of the unexpectedly (at least, unexpected from an evolutionary perspective) high levels of LD.

As expounded upon by Pritchard and Przeworski, 'To understand LD in humans, we need to have a much better understanding of human demography. This includes the history of changes in population size, as well as population structure and other forms of nonrandom mating.'[23] There is a very limited understanding of these issues, and new research will hopefully aid understanding of such complex genetic processes. Until then, models used to explain such evidence will remain highly theoretical.

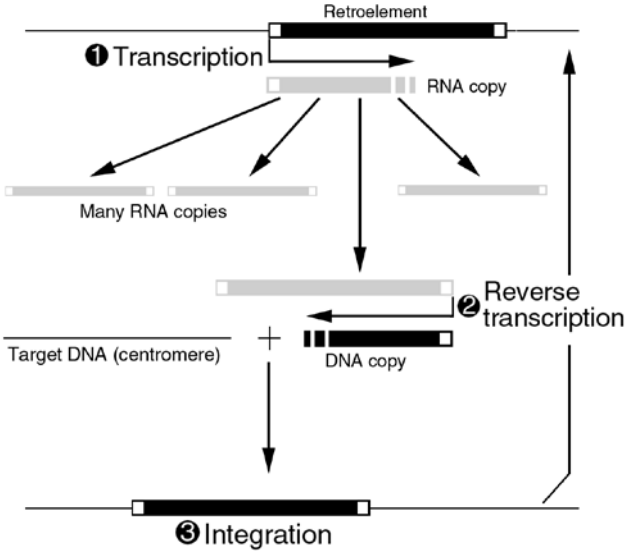
Out-of-Africa

Much evidence exists of the evolutionary Out-of-Africa hypothesis, the idea that modern humans arose in Africa ~200,000 years ago and spread out, replacing other more archaic humans.[24] Tishkoff *et al.* comment that 'the difference in haplotype variation at the CD4* locus* between sub-

Saharan African and non-African populations is striking and cannot easily be accounted for except by a recent common origin of non-Africans from Africa'.[16] Zhivotovsky *et al*.[25] also found that genetic variation increases within populations in the following order: the inhabitants of America, Oceania, East Asia, Eurasia and sub-Saharan Africa (with the most variation), hinting at an African origin.

The logic is simple: because Africans contain the greatest amount of genetic variation, and this in turn is taken to indicate a long population history, it is in Africa that our origin can be found. In the words of evolutionary anthropologist Mark Stoneking, 'Our genes contain the signature of an expansion from Africa within the past 150,000 years or so.'[26]

Although Reich *et al*.[19] uncovered evidence of long-range LD, this was specifically concerning Europeans. They also studied 96 Yorubans from Nigeria, and LD seemed to extend less far. It is significant to note, however, that very little of the LD is actually *specific* to the Yorubans. Also, the number of Europeans sampled was only 48, so bias did exist in the study.[27] Additionally, there is little evidence for major differences in the extent of LD among various European populations, indicating common ancestry.[14] In the context of evolution, the genetic evidence currently available is taken to indicate 'that there were only about 10,000 breeding individuals for a long time before the recent expansion of modern humans outside Africa'.[28] A population bottleneck is also suggested at 70,000 years ago, caused by the eruption of Mount Toba in Sumatra.[6] However, there has (to my knowledge) been no thought as to how this evidence for an African origin fits into biblical history. An attempt at a preliminary model follows.



Replication cycle of a retroelement—a type of transposable element or jumping gene—involving the copying of its RNA back into DNA. Transposable elements are segments of DNA which change position in the genome, and which could have played a role in regenerating rapid diversity after the Flood bottleneck by causing random variations (after Kidwell and Lisch).[54]

The Bible records a severe bottleneck ~4,400 years ago in mammals, birds and reptiles (Genesis 7, the Flood bottleneck) followed by rapid expansions (Genesis 9:1, God's command to multiply). After the Flood, numerous organisms would undoubtedly have struggled to survive or totally become extinct. This would have been followed by yet another bottleneck specific to *Homo sapiens* at Babel (Genesis 11) as languages were formed, and more expansion in humans would have occurred. Organisms would have been created with maximum genetic variability only ~6,000 years ago, and would thus still contain much more of this than assumed in a long-age model. According to this framework, the *least* genetic diversity should be found within humans (due to the Babel event), with greater diversity in mammals, birds and reptiles, and the greatest diversity in fish, amphibians, etc. (which were not destroyed as completely as land animals by the Flood). Additionally, organisms with shorter generation times would be expected to be more diverse, as heritable mutations occur between generations.

Rapid generation of diversity and 'junk' DNA

Adaptive radiation could have occurred in many species, potentially compensating for some of the diversity which was lost. Indeed, it has been noted by Heyer *et al.* that 'In a simple model of stationary population followed by demographic expansion, most of the mutations will occur during the expansion phase.'[29] Surtees[30] suggests that conditions directly following the Flood would have preserved all alleles which survived, and that any new alleles would have become fixed. He also describes many mechanisms by which genetic diversity could have been regenerated, including (but not limited to) accelerated point mutations, functional processed pseudogenes, exon shuffling and DNA translocations.

A great deal of recent research has supported the idea that it would not be as difficult as previously assumed to acquire the diversity seen today since the Flood. Gibbs notes that

> 'a genome [the entire DNA content of an animal's cell] is a biochemical machine of awesome complexity. Like all machines, it operates in three dimensional space, and it has distinct and dynamic interacting parts.'[31]

One such part includes the world of transposable elements. Also called jumping genes, transposable elements are segments of DNA which actually change position in the genome, either by moving to a location on the same chromosome, or to another one altogether. These genetic elements could have played a role in regenerating rapid diversity after the Flood bottleneck by causing random variations.[32,33] Walkup[34] notes that God could easily have designed such elements to move about or recombine in the genomes of organisms, allowing the rapid diversification seen in the 500 years or so after the Flood. It is also possible

that, under the somewhat crowded conditions of the Ark, a significant amount of genetic material was transferred between organisms through viruses, parasitic mites, or fleas. This is supported by new research which indicates that horizontal gene transfers have occurred between the protozoan *Trypanosoma cruzi* and its hosts, identifying a total of integration sites in the patients studied. Rabbit and human data also show that ß-globin loci and LINE-1's* are frequent targets for integration. It was concluded that this information 'might be more important for eukaryotic evolution than we previously realized'.[35] Of course, discarding evolutionary assumptions, it could just as easily be said that this plays a significant role in post-Flood diversification.

Additional evidence exists that harsh environmental conditions can induce mutations. For example, some evolutionists have proposed that enzymes such as DinB have evolved for the express purpose of producing genome-wide genetic variation under stressful conditions, allowing organisms to adapt rapidly.[36] Bishop and Schiestl note that homologous recombination, which can produce extreme variability,[21] can be stimulated by environmental carcinogens, though 'loss of genetic information' is still 'a common event'.[37]

It is also true that the genome is extremely complex, in that it contains many more parts than just protein-coding genes. One such part includes the introns, which are segments of DNA lying between other DNA segments that code for protein. When DNA is transcribed, the introns are spliced out (deleted). Because of this, they have long been assumed to lack function. However, if this is in fact the case, why have they been preserved in genomes? Indeed, as noted in *Scientific American*, the preservation of introns through time 'suggests they do something indispensable'.[38] It is possible that these DNA sequences, usually said to be worthless 'junk', may actually function as 'islands' of DNA which encode non-coding RNA (ncRNA). This ncRNA is made from DNA, but does not go on to become translated into protein: instead, it patrols the nucleus and acts as a regulator of other genes. This function is carried out when the ncRNA inhibits a complementary strand of messenger RNA (mRNA) by binding to it, thus preventing its translation into protein and rendering it inoperative.

In fact, it has even been suggested that differences in gene *expression* due to ncRNA, rather than differences in genes themselves, may actually be 'the primary cause of the biological differences between humans and chimps'.[39] This statement was echoed in *Scientific American*, which stated that 'Some scientists now suspect that much of what makes one person, and one species, different from the next are variations in the gems hidden within our "junk" DNA.'[38] Evolutionary assumptions about junk DNA have failed miserably. Mattick notes that 'when their truth cannot be immediately tested and their flaws are not obvious, assumptions often graduate to *articles of faith*, and new observations are forced to fit them' [emphasis added].[3] He goes on to explain that the secrets of this once-thought-junk DNA could be linked with several medical conditions, including B cell lymphoma, lung cancer, prostate cancer, autism, and schizophrenia. This is an obvious example of evolutionary theories hindering important fields of applied science.

Pseudogenes have also been observed to play a role in gene regulation, both by encoding ncRNA and by producing short peptides that bind to normal proteins, rendering them inoperative.[40] Many have noted that it is very probable that widespread pseudogene function exists, despite so-called 'disabling lesions', which exist in pseudogenes with known functions. Even the classic test of a high non-synonymous/synonymous mutations ratio does not necessarily mean lack of function, as even indisputably functional genes can have such a ratio at, or approaching, 1.0.[41] It must be acknowledged that research has only scratched the surface of the dynamic nature of the genome. Consider some of Australia's wallabies, for example: even though massive genetic change was observed to occur, including extreme chromosomal disfiguration, the phenotype of the wallabies remained virtually identical.[42] This implies a major role for *epigenetic* factors in the preservation of the different kinds, totally apart from DNA.[38,43] Scientists certainly have a long way to go before an adequate understanding of the genome's role in heredity and diversity is reached. In addition to this discussion, Woodmorappe[44] has provided a comprehensive creationist response to criticisms regarding Noah's Ark and the Flood, including, but not limited to, the field of genetics.

Evidence to date seems consistent with the basic creation model. One question remains: why is there such diversity and so little LD in Africa? Before it is assumed that less LD does, in fact, exist in Africans, it should be noted that some evidence *does* conflict with this. For example, the southern Bantu exhibit more LD than other African population estimates.[45] It has often been argued that we do not have an African origin.[46,47] This data requires evaluation in a creation model.

Out-of-Babel

Great African diversity is, contrary to popular thought, quite explainable within a biblical framework. Ambrose has commented that

> '[African] diversity is widely assumed to be a function of the greater age of African populations. Genetic diversity is, however, also a function of population size. Relethford and Harpending note that diversity due to a large population size can be incorrectly interpreted as being due to greater population age.'[6]

Geneticist Alan Templeton also believes that this is a possibility, and has been cited as arguing that differences in such ancient population sizes could actually 'mimic' a sequence of events similar to that postulated by the Out-of-Africa hypothesis.[48]

Here again it can be seen that the same evidence may be *interpreted* many different ways. The fact that many evolutionists recognize the validity of the explanation which

Oil painting (1563) of the Tower of Babel—the historical event during which God confused the human language—by Pieter Bruegel (1525–1569).

is consistent with the Genesis 11 account (although without considering this account, and obviously without supporting the same *timescale*) is encouraging. Therefore, for an unknown reason, more people simply ventured towards Africa after Babel. Perhaps because they had come from the east, journeying west (Genesis 11:2), they sought to continue. Many would have thus ended up populating Africa. If this model is correct, and more humans settled in Africa, this larger population size could account for the greater genetic diversity found there. Admittedly, although this is consistent with biblical history, it was not directly predicted by it (given *only* the information presented in the Bible). It would certainly be a difficult task to guess where the majority of humans settled after Babel based only on the biblical account. However, although this is somewhat a rare case in which the information given in the Bible does not permit the making of a prediction one way or the other, it is still perfectly congruent with the information population genetics has offered. This possibility warrants research. Most specifically, however, the supposed greater diversity of Africans and the significance of this requires creationist attention.

As research progresses in this field—it is still too early to make any concrete conclusions—predictions of a biblical model can also be tested. For example, it can be predicted that other organisms will show genetic signs of a bottleneck—although not as severe as for humans (who experienced Babel in addition to, and more recently than, the Flood). Bottlenecks have been detected in African Cheetahs,[49] along with a bottleneck in chimpanzees synchronous with that of humans.[6] Evidence dealing with domestic dogs points to a common origin also.[50]

Although linguistics has provided much support for the event at Babel,[51,52] genetic evidence remains to be explored in as much detail. Even though many humans could have moved to Africa, we would still expect to see evidence of human spreading in all directions (which is also observed to some degree).

Conclusion

Unlike evolution, it is by way of *prediction* that the Bible may be affirmed. Evolution is a very malleable doctrine, and is capable of compensating for any evidence that is found:

> 'evolutionary ideology is bound to naturalism, and simply "accommodates" all evidence to fit reworked evolutionary models, thus showing that it is not falsifiable and therefore not scientific according to science philosopher Karl Popper's primary criterion.'[53]

Biblical models of history are not like this. The Bible makes real predictions which enable its truth to be tested. Although evolutionary theories can certainly be invented to *accommodate* and *explain* the evidence for the bottlenecks observed, the information in the Bible has enabled this to be predicted from the beginning.

Acknowledgments

I wish to thank the following individuals, who collectively provided me with a great deal of commentary and/or information concerning earlier drafts of this manuscript: Jerry Bergman, Kim Risely, Ken Wadley, Tas Walker and Carl Wieland.

Glossary

Allele: Alternate forms of a gene or DNA sequence, which occur on either of two homologous chromosomes in a diploid organism.

Alu sequence: A type of short, non-mobile DNA sequence that occurs almost a million times in the human genome.

Autosomal: Belonging to any chromosome other than a sex chromosome.

CD4: Abbreviation of 'cluster of differentiation 4', and is a protein found on the surface of some immune cells.

Haplotype: A combination of alleles at two or more loci/positions in a region of a chromosome.

Haplogroups: A cluster or group of similar haplotypes.

LINE: Abbreviation of 'long interspersed element'. A mobile DNA sequence found highly repeated in eukayotic genomes, which reproduces itself through an RNA intermediate.

Locus: The position of a gene on a chromosome.

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