setting the limits to natural variation. Perhaps this was behind the Apostle Paul's statement that, 'not all flesh is alike, but there is one kind for men, another for animals, another for birds, and another for fish' (1 Corinthians 15:39). Differing cells can maintain differing ground-plans, while varying genes can allow adaptation to changing conditions.

Much work needs to be done to check out these ideas. But one thing is certainly clear—the Queensland wallabies have escaped from Weismann's paradigm.

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Genetics and Biblical demographic events

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With the relatively recent mapping of the human genome,[1] new questions can be raised concerning potential genetic evidence for Biblical events (specifically demographic events; that is, events affecting population) such as Creation and the global Flood. Evidence for a Mitochondrial Eve[2,3] suggests that the historical record in Genesis of one man and one woman at the beginning might be accurate, and this idea has already been discussed in the context of creation.[4] When actual measured mutation rates are used with the mitochondrial DNA data, the time frame for Mitochondrial Eve reduces to fit with the Biblical Eve.[5,6] Single nucleotide polymorphisms and linkage disequilibrium also provide relevant data concerning past populations, and could serve as quite objective evidence for such demographic events as a global flood, for instance. I outline a number of research findings and ideas here.

Genetic variation and the population bottleneck

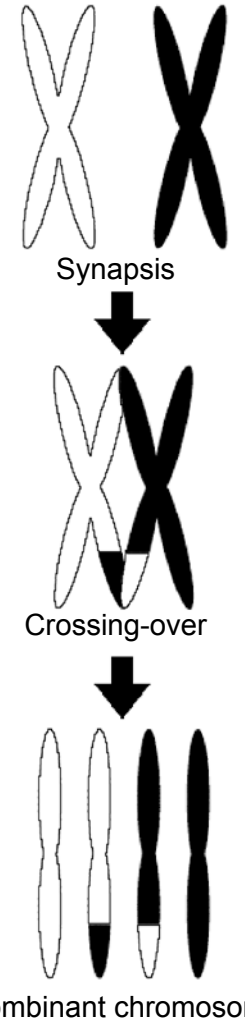
By comparing DNA from different humans around the world, it has been found that all humans share roughly 99.9% of their genetic material—they are almost completely identical, genetically.[7] This means that there is very little polymorphism, or variation. Much evidence of this genetic continuity has been found. For example, Dorit *et al*.[8] examined a 729-base pair intron (the DNA in the genome that is *not* read to make proteins) from a worldwide sample of 38 human males and reported *no* sequence variation. This sort of invariance

> '… likely results from either a recent selective sweep, a recent origin for modern *Homo sapiens*, recurrent male population bottlenecks, or historically small effective male population sizes … any value of Q [lowest actual human sequence diversity] $\geq$ 0.0011 predicts polymorphism in our sample [and yet none was found] … . The critical value for this study thus falls below most, but not all, available estimates, thus suggesting that the lack of polymorphism at *ZFY* [a locus, or location] is not due to chance.'

After citing additional evidence of low variation on the Y chromosome, they note in their last paragraph that their results 'are not compatible with most multiregional models for the origin of modern humans.' Knight *et al.*[9] have had similar research results:

> 'We obtained over 55 kilobases of sequence from three autosomal loci encompassing *Alu* repeats for representatives of diverse human populations as well as orthologous sequences for other hominoid species at one of these loci. Nucleotide diversity was exceedingly low. Most individuals and populations were identical. Only a single nucleotide difference distinguished presumed ancestral alleles from decendants. These results differ from those expected if alleles from divergent archaic populations were maintained through multiregional continuity. The observed virtual lack of sequence polymorphism is the signature of a recent single origin for modern humans, with general replacement of archaic populations.'

These results are quite consistent with a recent human origin and a global flood. Evolutionary models of origins did not predict such low human genetic diversity. Mutations should have produced much more diversity than 0.1% over millions of years. And yet this is exactly what we would expect to find if all humans were closely related and experienced a relatively recent event in which only a few survived. Research is needed to determine what variation should actually be present in the human genome—what would we *expect* within an evolutionary frame-



Crossing over (or genetic recombination) during meiosis, results in shuffled genes.

work, and how does that compare with what we find? These results could have a great impact on biological evolution, population genetics, and could provide telling results about the age of the humankind. It could also affect the so-called molecular clock.

Another piece of evidence involves single nucleotide polymorphisms (hereafter SNPs), which are mutations common to the human genome (meaning that many humans share them), being present in the human population at a frequency of roughly 1%.[7] These provide great insight into both medical research and population genetics. Many humans share large blocks of SNPs (called haplotypes), suggesting that all humans could have decended from a relatively recent demographic event.

Linkage disequilibrium (or LD) supports the same conlusion. Genes are located on chromosomes in cells, and these genes may either be far away or close to each other. All of the genes that are located on one chromosome are said to be linked. When cells divide through meiosis, crossing over (or genetic recombination) often occurs. This involves two chromosomes aligning and swapping segments of DNA, resulting in genes getting shuffled around.

The closer two genes are together, the more likely 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, genes are said to be in linkage disequilibrium—a state where they are not thoroughly mixed, but tend to be inherited together.[10]

Likewise, if genes *are* thoroughly mixed, they are in equilibrium. LD has provided much evidence for a population bottleneck, because humans contain long-range LD, or LD that extends quite far in the genome, meaning that many genes tend to be inherited together. This type of evidence has been found in Northern Europe, for example. In fact, data gathered by Reich *et al.*,[11] suggests that in general, blocks of LD are large in humans, because many genes are closely associated. The explanation of this can have significant implications:

> 'Why does LD extend so far? LD around an allele [or variant form of a gene] arises because of selection or population history—a small population size, genetic drift or population mixture—and decays owing to recombination [crossing over], which breaks down ancestral haplotypes [blocks of SNPs]. 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 [such as what we find in humans] 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.'[11]

This study concluded with the possibility that 50 individuals may have founded the entire population of Europe. This evidence is also quite consistent with a historical global flood. Research is needed on the implications of this data for the flood of Genesis. Certainly, humankind has undergone a relatively recent (tens of thousands of years at most, within an evolutionary time frame) population bottleneck. However, it must be further investigated as to the proportionality of evolutionary dates to the creation model, and as to how the molecular clock can be adequately explained in such a context. Data aiding this understanding has already been published.[5]

We should also seek to understand genetic evidence in the context of the tower of Babel event. Evidence exists that, after the bottleneck, 'the [human] population rebounded in a series of separate, rapid expansions on different continents.'[12] This too seems consistent with Biblical events in Genesis 11. Surely, much research is needed to expand ideas about such genetic evidence to determine its consistency with the Bible and its inconsistency with, for example, the various evolutionary out-of-Africa models. Different models of human history are hotly debated in the scientific community today.[13]

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Life by chance? Studies on folate co-enzymes add weight to that impossibility

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Life depends on folate

It is evident from our knowledge of biochemistry and molecular biology that there is a universal requirement for folates. No wild organisms survive without folate; thus there is no evidence of any organism that started without folate dependence or has attained an ability to dispense with folate. All life, from carnivores to plants to bacteria, has this dependence. In humans, their absolute importance is noted by health authorities[1] which have issued notification of a recommended daily allowance (RDA). For pregnant women, the RDA is increased to account for the growth requirement of the developing embryo. Modern diets which frequently lack the vegetables that are high in folate are under this level, so producers of processed foods, such as breakfast cereals, provide additional folic acid to supplement their products. This supplementation is supported by the National Health and Medical Research Council of Australia recommendations.[2] With insufficient folate supplementation, there can be disastrous consequences for a child developing *in utero*. Most notable are neural tube defects such as *spina bifida*.

What are the roles of folates? Folate, also known as vitamin B9, is actually a complex collection of molecules that do not occur by chance. Folates are coenzymes that are required by apoenzymes (proteins) to make an enzyme. Enzymes play a critical role in the chemical reactions that take place in life. Without enzymes, there is no known way that biochemical reactions could occur. Their purpose is to lower the free energy required to produce a product or an intermediate in the reaction. The way in which enzymes perform such processes is extremely complex. For example, the process to simply reduce dihydrofolate (DHF) to tetrahydrofolate (THF) requires the addition of two hydrogens to DHF (see figure). This cannot happen spontaneously, so all cells have the enzyme dihydrofolate reductase (DHFR) to carry out this reaction. Some of the complexity of the reaction can be see in a web movie.[3] For this reaction to occur, DHFR itself requires the coenzyme nicotinamide adenine dinucleotide phosphate (NADPH), which becomes oxidised in the reaction to make THF. The oxidised $NADP^+$ can be recycled by the cell to regenerate NADPH.

Folates are cofactors (or coenzymes) in numerous reactions leading to the synthesis of numerous amino acids for protein synthesis and purines for DNA synthesis, indicating just how essential folates are. These folate cofactors comprise several specific variations of a basic structure. The variations include THF, formyl THF, formino THF, methyl THF and methylene THF, and each are co-factors of a specific enzyme. In addition, because of the inherent instability of these compounds, they invariably have several glutamate residues added to them so they become impermeable to the cell membranes.[4] Thus if they are required to cross a membrane to be taken up by a cell or to be used within a sub-cellular compartment, the poly-glutamate has to be removed and then added again after the membrane has been crossed.[5] This is an essential process, and a specific enzyme is required to detach the glutamates, while another enzyme is required to add them on.

Fast growing cells have very high requirements for folates, and without them, cell growth does not occur. How so many cell types from such a range of organisms all depend on the same folate cofactors for these reactions is a testament to the fact that no intermediate life forms occur. It is easier to accept that all life was made to utilize folate in the same way.