of mutation does not result in an information gain, as Darwinism requires, but an information loss (often of a complete structure or protein). A chief difficulty in arguing for macroevolution by mutations is the fact that most expressed mutations are either lethal or semi-lethal. Either they kill the organism outright, or they prove harmful, so that in the ordinary course of life they are eliminated. This includes both mutations in which the fertility rate is reduced as well as mutations that result in the loss of certain structures.

And as shown, even the rare 'beneficial' mutation, as some might consider the Ancon to be, are the result of information *loss*. Therefore they are going in the opposite direction from what goo-to-you evolution requires.[14]

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Jumping paradigms

Alexander R. Williams

The paradigm that has ruled cell biology for more than a hundred years is under threat from Queensland's (Australia) wallabies. And the outcome just may provide creationists with a theory that explains the integrity of the created kinds.

In 1893, German biologist August Weismann published his 'germplasm' theory of inheritance. This says that 'germplasm' is the substance of inheritance, and it is transmitted independently of, and without interference from, the 'soma'—the body of the organism. Weismann's work refuted Lamarck's theory about characteristics acquired by the 'soma' being inherited. When the identity of the 'germplasm' was subsequently revealed to be DNA, Weismann's theory was turned into the 'central dogma' of molecular biology, which said that information could pass from the DNA to the cell, but not *vice versa*.

Although the 'central dogma' has been modified by the discovery of 'jumping genes' it still remains the major paradigm. Virtually all cell biologists today would believe that 'genes control cells'. One of the implications of this view is that there must be limits to genetic change, in order to maintain the integrity and viability of the cell. Experience with mutations shows that too much change can be fatal. If a genome were to become 'scrambled', 'completely haywire' and 'out of control' the cell should self-destruct. If 'very extreme and quite shocking' disfigurement occurred to the genome then drastic effects should result in the organism. If the equivalent of 'fifty million years of evolutionary change' (in evolution-speak) were to occur in 'five minutes' the impact on the organism should be catastrophic.

Well, apparently the rock hopper wallabies of the Queensland coast don't know this. Their genomes have suffered in exactly these ways yet the average person would think that nothing at all had happened to them!

Rock hoppers

Rock wallabies live on rock outcrops and cliff faces. Seven species occur widely scattered across Australia, mostly in geographically isolated populations, and they have distinctive colourations that tell them apart. But along the Queensland coast, another eight species live shoulder to shoulder in a linear geographic series, and they are so similar that only 'perhaps four people in the world'[1] could tell them apart by looks alone. It was not until genetic studies were carried out that the separate species were recognised.

Now in most animals the number and kind of chromosomes remains very stable. Any change to their number or structure is usually deleterious or fatal. But in macropods (wallabies and kangaroos), there is 'a tendency to play Lego® with their chromosomes, and in rock wallabies it's just gone completely haywire.'[1]

Recent studies were prompted by the curious case of 'Benny', a hybrid between two different species—the tall swamp wallaby and the tubby tammar wallaby.[2] Benny's chromosomes were found to have been seriously disfigured. Some of the centromeres (the place on the chromosome where the pairs join up) were ten times as long as normal; part of an arm of chromosome 2 had been moved to chromosome 7, and part of the X chromosome had been reversed. Analysis of Benny's DNA showed that it was 'dramatically under-methylated'. Methylation of DNA is a major method of controlling gene expression, so 'dramatically under-methylated DNA' means DNA that is 'out of control'. The researcher involved called it 'very extreme, and quite shocking'.[1]

When Benny's chromosomes with long centromeres were analysed they found pieces of retrovirus DNA repeated thousands and thousands of times. Retroviruses can insert themselves into a host's chromosomes, and on occasions may take with them a piece of the host's DNA, producing the phenomenon called 'jumping genes'.[2] The researchers suggested that perhaps

the hybridization might have caused the under-methylation, and the consequent breakdown of genetic control allowed the retrovirus to wreak havoc.

Benny's experience led to the idea that perhaps a similar mechanism had influenced wallaby speciation. A follow-up study of the Queensland rock hoppers turned up trumps. Dramatic changes of a similar kind were found, leading to an equally dramatic conclusion—'something that we thought might take 50 million years might take 5 minutes instead'.[1] From a conventional (evolutionary) point of view 'judging by their chromosomes alone, the eight species of Queensland rock hoppers look as if they diverged from one another 100 million years ago'.[1]

Something is wrong

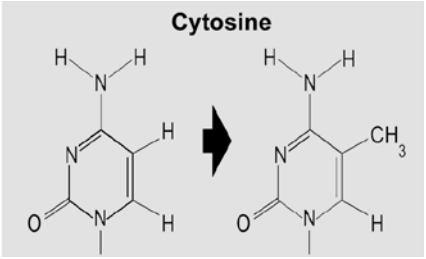
Something is terribly wrong here. Evolutionists cannot simply shrug their shoulders at these results. Weismann's paradigm cannot explain it. The genome in the rock wallabies has become 'scrambled' and 'out of control' yet only four people in the world can discern the morphological outcome. The DNA methylation system has suffered massive failure and caused the genome to go 'haywire', yet without noticeable effect on the organisms. The chromosomes have become as different as it is possible to be and still remain in the mammal class (i.e. the 100 million year time frame in evolution-speak) yet to look at them, you and I would say they are all the same species. The genetic change is 'very extreme, and quite shocking' yet the only impact on the wallabies is that the different species appear to prefer their own company.

Time for a paradigm change. What is the alternative? Well, if the genes don't control the cells then perhaps the cells control the genes.

Evidence for somatic control

The evidence for somatic control of heredity is impressive and growing. It seems that, in their zeal to find mechanisms to explain variation, evolutionists have overlooked the powerful structures supporting stasis.

- The fossil record and breeding experiments show that the dominant feature of life is stasis, not change. The structure that does not change during reproduction is the cell. When organisms reproduce, they pass on a whole cell to their offspring.
- Critics of neo-Darwinism long ago pointed out that 'oocyte cytoplasm is … a carrier of heredity independently of nuclear genes' and that subsequent development is dominated by epigenetic factors.[3] But it was only as late as 1992 that cell biologists began to realise that the 'cytosol' was in fact an incredibly complex structure[4] and it is transmitted unchanged from parent to daughter cell.
- Cell walls have very complex structural components that are passed on intact from mother to daughter.
- Organelles such as mitochondria, ribosomes and endoplasmic reticulum pass unchanged from parent to offspring. While certain nuclear gene mutations can (incompletely) suppress organelle development, organelles cannot be created *de novo* from genes—they must be inherited directly from their parent .[5]
- DNA does not read itself—ribosomes are needed for this. At cell division, countless ribosomes are passed from the parent to the daughter cell so the translation mechanism is passed on unchanged.
- It seems that, not only is the genetic code translated into protein by the cell, but the cell consults the nucleus only when it requires information.[6]
- Not all genes are active all the time. The decision as to which genes are active, and when they are active, is under epigenetic control—that is, the cell does it. It is a very complex process that has only recently become the subject of attention. Early results suggest that it can involve chromatin structure and histones[7] (proteins that surround the DNA and package it into the chromosomes) and non-coding RNA[8] (introns that are spliced out of the sequences that come from the process of transcription). In many, but not all organisms this control is exercised by the process of methylation, which is controlled by the cell.[9]



In vertebrates, DNA methylation (the addition of a methyl group) occurs at Cytosine nucleotides that are followed by Guanine. i.e. …CG…. Methylation is a control mechanism that turns off gene expression.

Created kinds

These results have an exciting bearing on the contest between the Darwinian Tree model of phylogeny versus the Creationist Forest model. Genesis biology requires integrity of the created kinds but also very rapid speciation to account for post-Flood biogeography. A Genesis genome must therefore have two fundamental components—a primary structure that maintains the integrity of the kind, and a secondary mechanism that allows for very rapid, but limited, variation.

Darwin expected natural variation to be continuous and unlimited. The extraordinary variability of genomes at the chromosomal and sequence levels gives some support to his view. However, while genomes do appear to be able to vary greatly, organisms *do not*. The Queensland wallabies are telling us that even the most extreme genetic changes can have almost *no* effect on the organism. Some force other than genetics must be at work.

The 'somatic' theory provides us with an explanation. If the control centre (or network) is in the cell and the created kinds have *different cells* then here we have a mechanism for

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

J. Warren Nelson

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] ≥ 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-