past, as Humphreys has inferred from data,[17] that would help to direct and concentrate charged particles towards the polar regions.

Summary

Although He-3 is formed in a different way than He-4, there are no reasons to believe that the abundance of this isotope in the lunar regolith is a major problem for creation science, and indeed *measured* lunar concentrations of He-3 are significantly less than the possible solar fluence over 6,000 years. By using average values and estimates from the solar wind and ignoring additional contributions, while seeking to minimise losses, there is found to be sufficient time to account for the concentration of helium-3 in the lunar regolith. Contributions from flare related and high-energy particle events are also likely to have a major impact, the historic frequency of which is unknown.

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Meiotic recombination—designed for inducing genomic change

Jean K. Lightner

Creationary biologists have recognized that the diversity seen within created kinds today cannot be adequately explained by the shuffling of pre-existing gene versions (alleles) and accidental errors that accumulate within the genome.[1] Within the context of creation, the development of genetic diversity has been a means by which God has enabled his creatures to adapt to the many different environmental niches they occupy today (Genesis 1:22; 8:17; Isaiah 45:18). Further, it has played an important role in adding variety, beauty, and productivity in various domesticated plants and animals.[2]

There is certainly no logical reason to believe that unguided chance processes can bring about a functional genome.[3] Neither is there sound reason to believe that accidental changes to the genome are a productive source of useful genetic diversity. Logically, therefore, the genome must contain biological information that allows it to induce variation from within.[4] One mechanism involved in this is meiotic recombination.[5] Continued scientific research is elucidating some amazing details of this process.

Meiosis is a special type of cell division necessary for the formation gametes (eggs or sperm) so sexual reproduction can take place. In most plants and animals, chromosomes come in pairs (homologs, one derived from each parent), but gametes only carry one of each homolog. Early in meiosis, each chromosome must be

drawn to its homolog and stably pair. Then each homolog will be pulled in the opposite direction so that the two cells that form during the division will have exactly one of each homolog.

Meiotic recombination is no accident

God designed meiosis in a way that naturally tends to increase diversity. In order for the chromosomes to stably pair, recombination occurs between the homologs. The process is initiated by an enzyme which cuts the DNA on one homolog, forming a double-stranded break. Then each side of the break is resected in one direction. This leaves two tails, which are important in repairing the break (figure 1).

There are several pathways by which the break can be repaired. The best known resolution of the break is called crossing over. For this to occur, both of the tails must invade the homolog to form a double Holliday junction (dHJ). DNA synthesis occurs extending these tails. Then, depending on which enzymes are used to cut this structure apart, the distal ends of the chromosomes are swapped. This swapping between homologs is important in helping to shuffle alleles, which allows for new combinations that may be advantageous.

The method of DNA repair described above is known as double-stranded break repair (DSBR). It does not always result in crossing over. A different enzyme can be used to cut the dHJ at a different location and gene conversion will result instead. In gene conversion, a segment from one homolog is copied onto the other. A second pathway for resolving double-stranded breaks is called synthesis-dependent strand annealing (SDSA). In this circumstance only one tail invades the intact homolog and gene conversion is the result.[6]

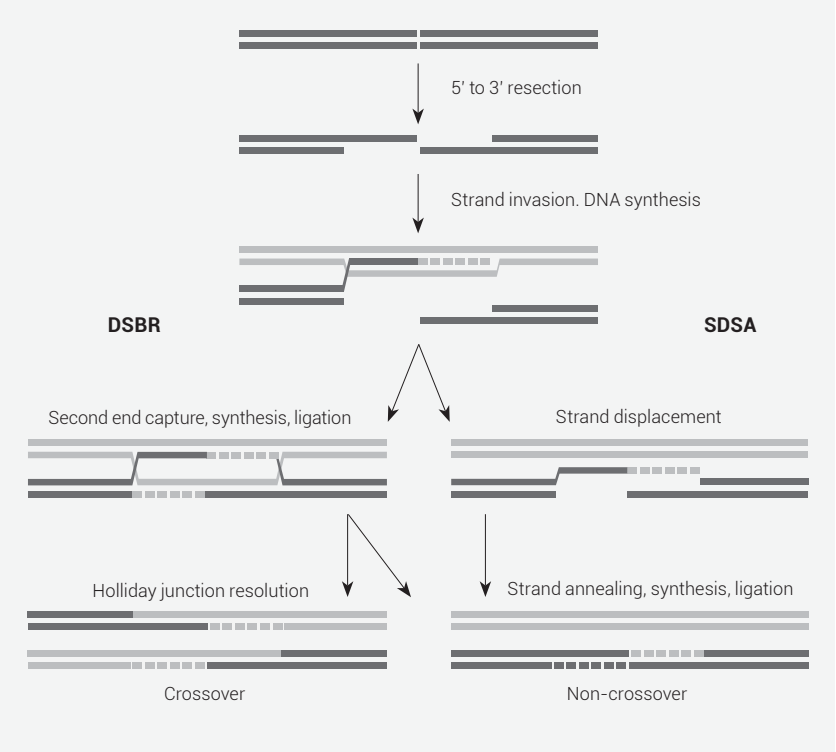

Figure 1. In meiotic recombination a double-stranded break is enzymatically induced and the ends are resected, forming tails. Repair of the break begins when one tail invades the corresponding region on the homolog and DNA synthesis takes place. From there several different pathways are possible. Crossing over can occur if the second tail also invades and a double Holliday junction forms. This pathway is called double-stranded break repair (DSBR). However, this pathway can have an alternative resolution, gene conversion (non-cross over). A second pathway is synthesis-dependent strand annealing (SDSA), which can also result in gene conversion.

Meiotic recombination is mutagenic

Technically, swapping portions of a chromosome and gene conversion are mutations when they alter the nucleotide sequence. Other mutations can also occur during the repair of double-stranded breaks. It appears to be more common with gene conversion. One study in yeast revealed a mutation rate 1,000 times higher during gene conversion than the normal spontaneous mutation rate for that locus. Most mutations were base pair substitutions. About 40% of the mutations were attributable to some form of template switching. In yeast strains with a proofreading defect in a DNA polymerase, template switch mutations were absent.[7] This suggests that template switching is a complex, enzyme-driven process.

There is a bias to where meiotic recombination occurs. In a study of *Drosophila*, crossing over tended to occur in specific hot spots, but these were not influenced by whether or not it was a genic region. Gene conversion had a more uniform distribution, was more common among genic sequences, and was seen where crossing over was rare or absent. The authors emphasized the importance of having information on rates of recombination to include in population genetics models.[8] Studies in plants indicate that a variety of genetic and epigenetic factors influence the frequency of crossing over.[9]

There are several other pathways by which double-stranded breaks can be repaired. One of the most interesting and mutagenic is break-induced replication (BIR). It has been shown to produce complex rearrangements including

copy number variation (CNV) and non-reciprocal translocations. These often involve multiple rounds of template switching. Specific endonucleases are necessary for proper BIR; an absence of these endonucleases has been shown to significantly reduce template switching.[10]

Significance of mutations

At times mutations are explained as the result of accidents which introduce errors into the DNA sequence. The concept of non-directed change is foundational in the standard evolutionary model. Logically, accidental changes in a complex system should be consistently harmful to some degree. Creationists have pointed this out in emphasizing the implausibility of accidents in accounting for the complexity of life.

However, when diversity is examined within a creation model, it is evident that significant diversity has arisen since the time of the Flood. In contrast to the notion that all mutations are harmful, the observed diversity does not appear to be typically harmful, and much is considered to be healthy. It has been pointed out that this useful diversity is not logically the result of accidents, but some designed mechanism(s) must be producing it.[1]

Several specific examples are worth noting. In a gene influencing coat colour, a pattern of in-frame indel (insertion or deletion) mutations was noted across several unrelated kinds. These generally result in a black coat colour. Statistically, only one in three indels should be in-frame. It does not appear that natural selection can explain this bias toward in-frame indels, and so a designed mechanism was suggested as its source.[11]

Resistance to organophosporous insecticides has been studied in sheep blowflies. There is a particular gene where specific mutations can confer resistance to one organophosphate or another. Resistance to one of these insecticides (malathion) was identified in pinned specimens that pre-dated the first use of that insecticide; therefore, selection would be a reasonable explanation for how it spread in the fly population. Resistance to a second insecticide (diazinon) appears to have arisen by mutation since the insecticide was introduced. This rapid appearance of resistance is quite impressive (though disheartening for those trying to get rid of this pest). In addition to this, flies have emerged that are resistant to both insecticides as a result of gene duplication (a form of CNV). It appears that such gene duplications have arisen at least three separate times in these flies, and always involve the resistant alleles.[12]

The point here is that the mutagenic nature of meiosis appears to provide a plausible mechanism for inducing this type of variation within a creationary timeframe. The requirement of specific enzymes and non-random pattern of change in meiotic recombination suggests it could play a significant role in producing the observed useful genetic diversity.

Gene conversion, a designed mechanism which can result in fixation of alleles

Gene conversion can lead to a transmission distortion, a deviation from the expected ratio of alleles in the gametes. Studies in mice revealed an example of this due to a preferential induction of double-stranded breaks on one homolog, which yielded an over-transmission of the allele from the other. Given the distortion, population simulations predicted that the favoured allele would be fixed in the population in less than 1,200 generations.[6]

Transmission distortion is extremely significant. Most models attempting to explain the changes in allele frequency of a population assume that a heterozygous parent would have an equal chance of passing either of the alleles on to the offspring. The fixation of alleles within a population is generally attributed to natural selection, although genetic drift is also recognized as a possibility. These are naturalistic explanations that fit well within the 'anti-designer' presuppositions of the evolutionary model.

Despite the appeal of scenarios crediting natural selection, they may have little semblance to reality if designed mechanisms are involved in changing allele frequencies. One example in animals would be migration. Perhaps animals move to where they are most comfortable because God gave them the wisdom to do so, thus enabling them to survive and reproduce. This comfort factor may be related to having a genotype compatible with (adapted to) that environment. So essentially, animals with adaptive alleles stay, and the others leave. This is rather the reverse of natural selection (where the environment 'selects' the animals), as it is the animal making a conscious choice.

Transmission distortion due to gene conversion, as described above in mice, may also prove to be an important mechanism for fixation of adaptive alleles in populations. If this turns out to be the case, it is a serious problem for evolutionists. It would be another major blow to the view that naturalistic processes adequately explain the origin of new species. Instead, designed mechanisms would be important for both the generation of diversity and fixing adaptive alleles within a population. If designed processes are necessary for adaptive changes even within created kinds, it points again to an awesome Creator!

Summary

One thing is clear; the evolutionary based inference that mutations (any change in the DNA sequence) are always accidents or copying errors is false. Changes in DNA sequence can arise for a number of reasons. One reason is that meiotic recombination, an essential step in reproduction for many plants and animals, is designed

to induce genetic changes. This is highlighted by the fact that enzymes are necessary for this complex processes, including enzymes which induce the double-stranded breaks and facilitate template switching. Since this is the case, I fully expect that better understanding meiotic recombination will be one piece in the puzzle to better understanding how diversity has risen so quickly within created kinds since the time of the Flood.

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DNA and bone cells found in dinosaur bone

Jonathan Sarfati

For the last 15 years, Dr Mary Schweitzer has been rocking the evolutionary/uniformitarian world with discoveries of soft tissue in dinosaur bones.[1] These discoveries have included blood cells, blood vessels, and proteins such as collagen. But under *measured* rates of decomposition, they could not have lasted for the presumed 65 million years (Ma) since dinosaur extinction, even if they had been kept at freezing point (never mind the much warmer climate proposed for the dinosaurs). Specifically, Buckley *et al.* measured the half-life of collagen at 7.5°C to 130 thousand years (ka). This measurement has been reliably repeated many times, so it represents the optimal conditions for molecular longevity at that temperature.[2] Schweitzer said in the popular TV show NOVA,

> "When you think about it, the laws of chemistry and biology and everything else that we know say that it should be gone, it should be degraded completely."[3]

She similarly noted in the journal *Science*:

> "The presence of original molecular components is not predicted for fossils older than a million years, and the discovery of collagen in this well-preserved dinosaur supports the use of actualistic conditions to formulate molecular degradation rates and models, rather than relying on theoretical or experimental extrapolations derived from conditions that do not occur in nature.[4]"

A careful scientist, Schweitzer rechecked the elastic blood vessels and other soft tissue, saying,

> "It was totally shocking. I didn't believe it until we'd done it 17 times."[5]

Other evolutionists saw the baneful implications to their long-age dogma, and claimed in 2008 that the blood vessels were really bacterial biofilms, and the blood cells were iron-rich spheres called framboids.[6] Yet this ignores the wide range of evidence Schweitzer adduced, and she has answered this claim in detail.[7,8] For example, independent labs have identified by antibody blot reaction and have even sequenced non-bacterial, vertebrate-specific proteins including collagen, elastin, osteocalcin, and laminin.[9] However, Schweitzer herself maintains her faith in the long-age paradigm.[10]

Dino bone cells and proteins

Schweitzer's more recent research makes long ages even harder to believe. Here, she analyzed bone from two dinosaurs, the famous *Tyrannosaurus rex* (MOR 1125;[11] figure 1) and a large duck-billed dinosaur called *Brachylophosaurus canadensis* (MOR 2598).[12] Bone is an amazing tissue, having the ability to rework in response to stress,[13] and it uses the finely designed protein osteocalcin,[14] which has been found in the best known duck-billed dinosaur, *Iguanadon*, 'dated' to 120 Ma.[15] The most plentiful cells in bones are *osteocytes*. These have a distinctive branching structure that connects to other osteocytes, and have a "vital role" in "immediate responses to changing stresses."[11]

Schweitzer's team again removed the hard, bony mineral with the chelating agent EDTA. They found "transparent cell-like microstructures with dentritic [branching, just the shape expected for osteocytes] processes, some containing internal contents", from both dinos.

They also used antibodies to detect the globular proteins actin and tubulin, used to make filaments and tubes in *vertebrates*. The proteins from both dinosaurs had similar binding patterns to the same proteins from