Can recombination produce new genetic information?

C. W. Nelson

It has been claimed that genetic recombination is able to produce new 'proteins with more complex functions', i.e. new genetic information.[1] Upon examination, the evidence used to support this claim proves weak. No evidence thus far has shown this claim to be true with respect to *hereditary* information, and most cases used are instances of an equivocal argument. When new information *is* produced, albeit in miniscule amounts, it is never heritable, and is produced via the work of complex cellular machinery that is clearly designed for the specific purpose of variability. B-cell maturation is not a relevant example when speaking of hereditary information, as the genetic rearrangements that take place in that process occur in somatic cells, not gametes, and are therefore not passed to offspring. Importantly, the creation of new alleles is *not* synonymous with the creation of novel protein functions. While it is, in principle, *possible* for new information to be produced, the odds against it are astronomically great, and there is no empirical evidence that it has happened. This proves to be another case in which the faith employed by the creationist is clearly equalled, if not surpassed, by the evolutionist.

In a recent issue of *TJ* (**18**(2)), Christopher W. Ashcraft published an interesting article regarding the role of homologous recombination in the production of genetic diversity.[1] I wish, first, to say that I found the paper very well written and thought-provoking, and I have certainly benefited from the information I gathered from it—I found that the raw data was sound. However, I also respectfully disagree with a number of points made. I do not believe that Ashcraft's ***conclusions*** regarding the creation of new proteins with more complex functions are justified by the data.

Genes (segments of DNA coding for a trait, such as hair colour) are located on chromosomes in cells. Chromosomes exist in pairs (except the sex chromosomes)—one from the mother and one from the father. Each parent thus contributes one half of the genes that affect the ***same*** traits in the offspring. Both genes, generally located at the exact same position on each chromatid, interact to determine the trait that will be expressed by the organism. For example, a human might contain a dominant gene for brown hair on one chromosome and a recessive gene for blonde on the other. Because the brown hair ***allele*** (or variant form of a gene) is dominant, the individual will grow brown hair.

Homologous recombination (hereafter HR) essentially swaps sets of genes between each homologous chromosome at meiosis (see figure 1). Therefore, rather than an organism being blessed (or burdened) with ***only*** one parent's genes, the offspring inherits a mix of both parents' traits. This certainly has the potential to produce great genetic variety in organisms.

Ashcraft accurately points out that many genes have more alleles than could possibly have been brought aboard the Ark. For example, the two members of the dog kind present on the Ark could have contained a ***maximum*** of four alleles for each gene (2 alleles x 2 individuals; recall that a different allele can be present on each of the homologous chromosomes). It is often claimed that the existence of more than four alleles in populations is proof that new genetic information has arisen since the Flood, and thus that evolution has occurred. Ashcraft, it seems, does not hold to the latter, but seems to agree with the former, stating that 'in spite of such concrete evidence, many creationists still tend to assume there is no mechanism for generating new genetic information'.[1] He then cites HR as a process which supposedly ***can*** give rise to new genetic information—namely, 'proteins with more complex functions'. I do not believe this is correct.

Examples of new information?

Ashcraft shows that genetic variability can be produced very quickly in populations in a directed manner; for example, following a ***population bottleneck***[2] (a severe reduction in the number of members in a population, followed by expansion). However, he makes the mistake of confusing the production of ***variant alleles*** with the production of new genetic information for the coding or more complex functions. He claims that ***gene conversion*** (a HR process that overprints one gene using another gene as a template) is actually responsible for such new information. At its conclusion, his paper cites another article, by Shibata ***et al.***,[3] which asserts that 'homologous recombination can use previously existing genes as building blocks, thus enabling the creation of new proteins with more complex functions in a step-by-step manner'. Let us review some of the examples given for this, beginning with those presented by Shibata ***et al.***

A closer look at the data behind the statement above, despite the claim that it is 'more than a hypothesis',[3] exposes it as a mere assertion. A study by Crameri ***et al.***[4] is cited to add weight to the idea. In the study, four related (that is, orthologous) 1.6 kilobase genes from different species were

fragmented and combined, analogous to HR. It was shown that in just a single cycle of such 'family shuffling' the rate of functional enzyme improvement accelerated 34- to 68-fold! This is certainly powerful evidence for HR as a possible mechanism of the rapid production of diversity, but does it provide an example of new information arising? No!

Note, first, that the ***previous*** function of the enzyme was improved, but a new function was not obtained. However, the latter is exactly what Shibata ***et al.*** claim the evidence proves. This argument resembles a classic equivocation, or bait-and-switch argument.

A fairly accurate analogy can be made by the following two sentences:

A. My friend Steve put his books in the boot of his car.
B. My mate Steve put his books in the trunk of his car.

Notice that each sentence has an identical meaning. However, the ***end*** of sentence **A** and ***beginning*** of sentence **B** contain words that would be better suited for an Australian reader. A 'recombination' event can effectively combine the appropriate halves of the two sentences in order to produce the best possible combination for our Aussie friend:

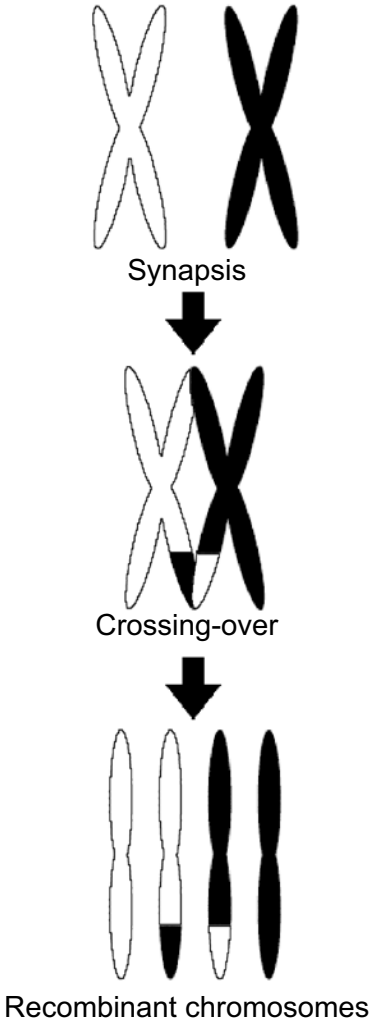

Figure 1. *Recombination during meiosis*

C. My mate Steve put his books in the boot of his car.

However, this sort of change is taken as an example of a sentence with an entirely ***new*** meaning being produced. Additionally, and unlike sentences, proteins contain specified amino acids that interact to drive very precise folding of the protein into a highly conserved shape.[5] Imagine, then, that a new requirement for our sentence includes the ability to 'fold' upon itself at the centre letter so that each letter can match its twin after the sentence has 'folded' (in case this seems confusing, I shall provide an example in a moment). Remember, too, that the sentence must also have intelligible meaning (it must be a relevant complete sentence). Because I haven't the ingenuity (or patience) to invent a phrase that fulfils these criteria, please join me in imagining that the phrase 'racecar level racecar' contains some profound, relevant meaning. This would work, then, because the phrase can accurately fold:

D.

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          v
        e - e
      l   -   l

    r     -     r
  a       -       a
c         -         c
  e       -       e
    c     -     c
      a   -   a
        r - r
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Although this is by no means a perfect analogy, I hope it effectively communicates my point: it has been ***claimed*** that a new sentence (fulfilling the requirements of **D**) can be produced by mixing sentence fragments, but only an example of the sort of rearrangement exhibited by **C** has been shown. In simpler words, this is yet another example of using selection (inaccurately) as evidence that information-gaining evolution has occurred.

It should also be noted that the genes used in the Crameri ***et al.*** study were orthologous genes thought to have arisen by ***convergence*** (similar genes having evolved separately, with no common ancestor). Whereas normal HR requires very high sequence similarity between the DNA segments involved (usually containing many genes), the exclusive rearrangement of allele segments of the ***same*** gene did not require as much similarity (58–82%). Such evidence could easily affirm the hypothesis that similar genes in separate species were created by a Designer for common functions, especially in light of the fact that evolutionists believe convergence produced them (that is, the similarities did not arise because of common ancestry). In effect, I believe that Ashcraft has been tricked by the mere assertions of evolutionists (who do, of course, genuinely believe that their example lines up with the assertions made). Rearrangements could surely have occurred in the genes of dogs (the example used a number of times by Ashcraft), and this would have generated diverse alleles, but it does ***not*** serve as an example of new genetic information. I am unaware of any other supposed examples of new information arising by means of HR, and thus I do not believe that Ashcraft's statement regarding creationists 'assuming' that no genetic information can arise, 'in spite of such concrete evidence', is sound. As stated by Spetner,[6] 'New DNA sequences [can] come only through mutations. Recombination can't do much more than bring out what's already there.'

What do creationists claim?

Upon reading the creationist literature, one will surely find that creationists do not reject that information ***can*** arise—the ***possibility*** of such an occurrence is present. They simply recognize that the astronomically small probabilities of such an event occurring render the neo-Darwinian theory useless. I believe most young-earth creationists would agree with Spetner when he states that 'The hard question that tests the validity of the theory is this: Is the chance of building up small [information-gaining] changes large enough to make the [neo-Darwinian] theory work?' and 'It is not impossible, in principle, for a mutation to add a little information, but it is improbable.'[7]

In the issue of ***TJ*** published two issues before Ashcraft's article, Dr Don Batten dealt with the adaptation of bacteria to feeding on nylon waste.[8] He states outright that

> 'The results so far clearly suggest that these adaptations did not come about by chance mutations, but by some designed mechanism. This mechanism might be analogous to the way that vertebrates rapidly generate novel effective antibodies with hypermutation in B-cell maturation, which does not lend credibility to the grand scheme of neo-Darwinian evolution.'

This is precisely what Ashcraft has shown and supported. What he did ***not*** support were his own statements that new proteins with more complex functions have been produced (which have relevance to ***heredity***). His claim that 'many in the creation science community have been denying the existence of new alleles rather than looking to cellular mechanisms as the source' is also utterly false. In fact, the very example he uses of the creation of new alleles has been commented on for the layperson by Wieland.[9]

B-cell maturation and such

An example provided is that of B-cell maturation. A B-cell, produced in bone marrow, contains antibodies on its surface that act as the 'fingers' of the immune system.[10] That is to say, the protein that makes up the binding sites (also known as the variable regions) has a precise shape which can fit a part of a bacterium or a virus. If the fit is just right, it sets off a complicated (and irreducibly complex) immune response that works to destroy the invader. The most common type of antibody is Y-shaped, with receptor sites at its tips (see figure 2). However, there is a problem: each B-cell produces antibodies with only one very specific binding site. If each B-cell had antibodies with identical receptor sites, only one 'shape' of invader could be protected against. Very well; the cell makes millions of B-cells, with millions of different receptor sites (hence, the ***variable*** region). These sites are created by combining, at random, DNA segments millions of bases apart to encode a unique protein during the B-cell's development. After the cell makes sure that this shape cannot actually fight the body's ***own*** tissue, it is released for its hunting.

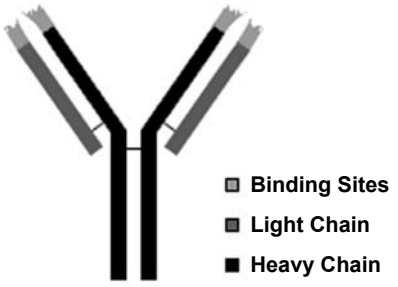

Figure 2. *A typical antibody molecule*

However, this is not an example of new information for a new function either (let alone new ***heritable*** information)! A rearrangement of ***pre-existing*** information is certainly involved, but combinations of this are not examples of diversity that can be inherited. Moreover, the same function is retained—that of 'kill the invader'! (Evolutionists have sometimes used B-cell maturation as an analogy for biological evolution, but for this same reason, it is irrelevant.[11]) Ashcraft accurately points out that there are precise cellular mechanisms ***designed*** to combine the information that is present in new ways. However, it seems he misses the important point that the rearranged sequences are ***not*** passed on to offspring, and so this argument is not relevant to the thesis: indeed, Truman[12] shows that it is the 'original, pristine gene fragments [that] are maintained in the germline lines of the vertebrate population'. The immune system is analogous to a robot, having been preprogrammed to 'find its way around a room'.[12]

Also, if information is relative to increases in ***specificity***, as Spetner[13] points out, a line of reasoning can be drawn where the level of specificity of every antibody produced is quite the same, relative to the same function.

Ashcraft also claims that recombination can occur in mitochondrial DNA (hereafter mtDNA, a ring of DNA found within the mitochondria of the cell), citing a short paper by Strauss:[14] 'Mixing of paternal with maternal mitochondria sequences was recently found, and the investigators have concluded that recombination occurred between the organelles following fertilization.'[1] However, the investigators did not ***conclude*** the issue at all, as there has been significant dispute over that article, and the text itself shows that other explanations besides recombination exist. Over and again, examples of mtDNA recombination have proven false,[15–17] with the possibility of slight paternal leakage.[17,18] There may be some newer evidence supporting recombination in mtDNA,[19,20] but the Strauss paper, as cited, certainly does not conclude the matter.

Rapid post-Flood speciation

HR most likely did play a significant role in rapid speciation after the Flood. Adaptive radiation could have occurred in many species, potentially compensating for some of the diversity that was lost. Indeed, it has been noted by Heyer ***et al.,*** regarding mtDNA, that 'In a simple model of stationary population followed by demographic

expansion, most of the mutations will occur during the expansion phase.'[21] Gibbs[22] 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.'

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 diversity after the Flood bottleneck by causing random variations.[23] Transpositional bursts are thought to be related to environmental stress,[24] which would certainly have been the case following the Flood. In fact, a recent article suggests that transposable elements actually have the ability to repair DNA.[25] Walkup[26] 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 could have been transferred between organisms through viruses, parasitic mites or fleas. Pleiotropy (and polygeny) should also be kept in mind when considering the origin of post-Flood variation.[27]

I would argue that this does not provide an example of new genetic information for ***novel protein functions***, and also that the examples cited by Ashcraft are irrelevant to heredity. Undoubtedly, HR may produce variation in offspring: but this has not yet been shown to add information to a genome.

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C. W. Nelson is a high school junior with a particular interest in biology as it relates to evolutionary theory. He often gives talks at his school's Fellowship of Christian athletes, on topics ranging from Genesis to Christian love. His current research interests involve population genetics, chemical evolution, and the works of C.S. Lewis.