# PERSPECTIVES ON AGEING, A YOUNG-EARTH CREATION DIVERSIFICATION MODEL

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**ABSTRACT**

The AGEing model proposes that intrabaraminic diversification occurred because of the action of transposable and repetitive elements, called Altruistic Genetic Elements (AGEs). Since the model was proposed in 1999, much new evidence has come to light, some of which supports the model and some of which calls for a significant revision of the model. Evidence of AGE/gene association, AGE horizontal transfer, and AGE-induced genetic changes all support the original AGEing model. Evidence of extensive genomic rearrangement, bacterial plasmids, and AGE transposition control requires substantial modification of the AGEing model. A new model of diversification based on these evidences will be introduced and explained.

## INTRODUCTION

Creationists have long accepted intrabaraminic diversification, and some agree that a burst of rapid diversification occurred during the first few centuries after the Flood [6,65,67]. Other creationists have been slow to acknowledge the challenges that arise from diversification. For example, a new report places all fossil equids in a single monobaramin, implying that they descended from a pair of horses on the Ark [12]. This would require the origin of 150 species of horse prior to the gift of donkeys (*Equus asinus*) from Pharaoh to Abram (Gen. 12:16), which took place ~400 years after the Flood. Similar examples of rapid diversification in other vertebrate baramins could be given (e.g. see Wood [68]). Since we see no 'speciation' like this today or in historical times, we can infer that the mechanism of intrabaraminic diversification is qualitatively different from modern speciation [68].

Despite the mechanistic difference between speciation and diversification, most creationists follow Marsh's lead in proposing micro-evolutionary or speciation processes to account for intrabaraminic diversification [39]. Marsh advocated a four-fold explanation of speciation within baramins encompassing recombination, gene mutations, chromosomal aberrations (including mutation and ploidy changes), and hybridization. Many modern creationists have adopted a Mendelian model of diversification in which a created gene pool is fragmented into smaller pieces containing less information (fewer alleles) than the original [52,66]. To address the need for a novel mechanism to explain diversification, I proposed a model called AGEing (for Altruistic Genetic Elements, see below) [68].

Since the proposal of the AGEing model at the Baraminology99 conference (Liberty University, Aug. 5-7, 1999, http://www.bryancore.org/bsg/meetings.html), much new evidence has come to light regarding the model. In this report, I will briefly review the AGEing model with particular emphasis on the research

applications of the model. I will then review the aforementioned new evidence. In light of this new evidence, I will expose some difficulties with the original model and illustrate how the model is a limited and specific example of a larger phenomenon, here called 'genomic modularity.'

## THE AGEING MODEL

The AGEing model begins with the common creationist belief that God placed the genetic information necessary for intrabaraminic diversification into the biosphere at creation [4,66]. In contrast to other creationist diversification theories, the AGEing model proposes a unique mechanism by which this genetic potential manifests itself. I proposed that transposable and repetitive elements originally functioned to activate latent genetic information or deactivate previously active genetic information for the purpose of effecting intrabaraminic diversification. Many evolutionists view these elements as "selfish DNA" that exists just to replicate [17,46]. To distinguish my model, I call transposable and repetitive elements Altruistic Genetic Elements (AGEs) [68]. I refer to intrabaraminic diversification mediated by AGEs as the AGEing process or the AGEing model.

I proposed several possible mechanisms by which AGEing could alter gene expression, and thereby induce novel phenotypes. First, AGE insertion into or excision from promoter regions could activate or inactivate adjacent genes. In some cases, the AGE itself could carry the promoter; in other cases the AGE could disrupt a pre-existing promoter [14,16]. Other AGEs could act as enhancers, altering expression of genes even at non-adjacent positions on the chromosome [41]. AGEs could also act as recombination sites, altering the natural chromosomal crossing over that occurs during meiosis [10]. Finally, AGEs could actually transport genes, promoters, or enhancers from one genome to another.

According to my original model, the key to AGE function is insertional specificity. AGEs inserting at random positions in the genome would induce changes at the same frequency as other mutations, yielding little benefit to the host organism. In order to facilitate rapid phenotypic diversity in just a few centuries, AGEs must transpose into specific genomic positions. Since I proposed that AGEs were designed by God to produce the diversification of their hosts, God must have designed each AGE with high specificity for insertion. As time progressed, inevitable mutations in the AGE sequence itself began to corrupt the beneficial activity of AGEs, leaving us today with detrimental, "selfish" mobile elements.

The AGEing model lends itself to numerous immediate research applications. For example, my original paper listed three testable predictions of the AGEing model [68]. First, the model predicts that two species that belong to the same baramin will exhibit differences in their AGEs, possibly corresponding to the phenotypic difference between the species. This prediction can be addressed by comparative genomics. For example, research suggests that all ~10,000 species of grass belong to a single holobaramin [69], and genomic studies have highlighted differences in transposable element content and position between different species of grass [38,13].

The AGEing model also predicts that some AGEs (but not necessarily all) should exhibit a tendency to insert near genes. Since some AGEs could act as non-adjacent transcriptional enhancers, those that do insert near genes probably act as promoters or promoter-disruptors. For some AGEs, this gene-association tendency can be provisionally demonstrated. The *Caenorhabditis* genome appears to show a bias of some AGEs to be located near genes [63], and Miniature Inverted-repeat Transposable Elements (MITEs) are well-known for inserting near genes [9,75].

Finally, the AGEing model predicts that the members of baramins living just after the Flood were more adaptable than extant members of the same baramin. More specifically, AGE activity, and therefore diversification, should decrease exponentially after the Flood. This prediction is the most difficult to verify because it requires an interdisciplinary approach between baraminology and paleontology. Research in horse baraminology provides preliminary confirmation of this prediction. The generation of horse species after the Flood appears to decrease with time [71].

## NEW EVIDENCE THAT SUPPORTS THE AGEING MODEL

Since the introduction of the AGEing model, new data have come to light that support the model. Technical advances in eukaryotic genome sequencing, illustrated by the publication of several rough draft genome sequences [24,43,74], have provided a plethora of raw sequence data upon which to conduct AGE-related research. Genome studies have uncovered additional evidence of AGE/gene association. Work on transposable elements has also revealed examples of horizontal transfer of AGEs

within and between baramins [34,73]. Finally, several reports illustrate examples of non-detrimental AGE-induced genetic changes, which complement numerous examples of harmful changes wrought by presumably degenerate AGE activity.

***AGE-Gene Association.*** The AGEing model predicts a tendency for some AGEs to be positioned adjacent to genes. In my original presentation of the AGEing model, I suggested that the genome of *Caenorhabditis elegans* provided limited support for this prediction [68]. Nematode-specific genes appear to be associated with repeat sequences on the *C. elegans* chromosomes [63]. New studies of the distribution of MITEs on *C. elegans* chromosomes have complicated this picture. The MITE *Cele14* is distributed in a similar manner to the repeats initially identified in the *C. elegans* genome report, but the MITE *Cele2* has precisely the inverse distribution [61]. Thus, *Cele14* appears to avoid *C. elegans* genes that are homologous to yeast genes, but *Cele2* prefers them. If the AGEing hypothesis is correct, we could infer that *Cele2* acts as a promoter or promoter-disruptor, while *Cele14* has another function (enhancer?).

A similar claim of AGE/gene association could be made for *Alu* elements in the human genome. *Alu* elements are short interspersed nuclear elements (SINEs) of approximately 300 nucleotides (nt). These elements were recently active, as evidenced by their polymorphism in modern human populations [50]. *Alu* elements are known to insert preferentially into gene-rich regions of the human genome [57], and the draft human genome sequence confirms this observation [35]. Some researchers even propose beneficial functions for *Alu* elements to explain their preference for genic regions [35,53], although others reject these proposals [8]. The close association of *Alu*s and genes supports the AGEing model. Furthermore, the human genetic diseases linked to *Alu* insertion [42,60] are consistent with a degenerating AGE function.

As a final example of AGE/gene association, grass MITEs show a clear bias for inserting near genes. In an early survey of rice genes, MITEs appeared frequently in the untranslated regions of publicly-available rice gene sequences, in contrast to *Arabidopsis* genes which showed no such bias [9]. A more comprehensive survey of rice sequence-tagged-connectors (STCs), representing 10% of the complete genome, demonstrated a similar MITE/gene insertional bias [38]. Recently, a study of the maize genome revealed that over 50% of the sequences adjacent to a *Heartbreaker* MITE were similar to known plant genes [38,75].

***AGE Horizontal Transfer.*** The most controversial proposal of the AGEing model was that occasional AGEs could transfer a functional copy of genetic material (gene, promoter, or enhancer) from one species to another, possibly crossing baraminic boundaries [68]. This proposal represents a challenge to the creationist because we must propose a method by which to detect such an event. Evolutionists infer horizontal transfer by phylogenetic methods, which might not be acceptable to the creationist. Distinguishing a historical instance of horizontal transfer from the created condition of a gene can be difficult for the creationist working from the published literature. For example, the description of the public human genome sequence draft included the provocative proposal that 227 genes had been acquired directly from bacteria [35], but a follow-up study showed that many of these genes were also found in other eukaryotic genomes [51]. The occurrence of “bacterial” genes in various eukaryotic organisms could be interpreted as multiple horizontal transfer events or as God’s intentional design.

In my original discussion, I cited two instances of purported horizontal transfer, both from *Drosophila* [28,15]. I accepted both of these examples because of the probable baraminic relationship of the *Drosophila* species, which lends support to the phylogenetic assumptions underlying these particular studies. A study by Yoshiyama et al. reveals an example of AGE transfer between parasitoid and host. A *mariner*-like element (MLE) in the parasitoid wasp *Ascogaster reticulatus* is not found in other wasps of the same genus; however, a sequence 97.6% identical to the wasp MLE is present in the moth *Adoxophyes honmai*. Yoshiyama et al. proposed that the close association of the host (*A. honmai*) and parasitoid (*A. reticulates*) provided opportunity for the MLE to transfer from one to the other. They speculated that a virus could have been the vector by which the MLE was transferred [73].

***AGE-Induced Genetic Changes.*** Obviously, the most important prediction of the AGEing model is that AGEs can induce beneficial genetic changes at a rate higher than random mutations. Consequently, demonstration of a beneficial genetic alteration that can be linked to an AGE insertion or excision would support the AGEing model. My original presentation of the AGEing model at Baraminology99 included no examples of beneficial or even neutral alterations induced by AGE activity. At the time, examples of AGE degradation producing abnormal gene regulation were much more common. Since my original

presentation, evidence of AGEs directly influencing gene expression with no harmful effect has been reported.

A study by Carcedo et al. has revealed that the transcription of the human annexin A5 gene is influenced by the presence of the long terminal repeat (LTR) of an endogenous retrovirus. Annexin 5 is a calcium channel protein that has been functionally linked to anticoagulation. By deleting the LTR immediately adjacent to the annexin A5 gene, Carcedo et al. demonstrated that the presence of the LTR reduces the transcriptional activity of the gene [11]. This study is a direct confirmation of the main prediction of the AGEing model that AGEs will induce changes in genetic regulation.

Two other examples provide evidence of AGEs influencing the biological activity of hormone receptors. In 1999, Kapitonov and Jurka reported that alternative splicing in the human leptin receptor gene was induced by an endogenous retrovirus called HERV-K10. Leptin receptor recognizes the human hormone leptin, which regulates weight and puberty. The human gene for leptin receptor contains a HERV-K10 sequence in the intron preceding the terminal exon, generating at least two mRNA and protein products. Both protein forms are present in human cells, but they are thought to differ in biological activity, particularly in cell signaling upon leptin binding [32]. In 2001, Hughes reported a similar case with the human gene for vascular endothelial growth factor receptor 3 (VEGFR-3). Vascular endothelial growth factors regulate growth and re-growth of blood vessels. As with the leptin receptor, a HERV element is present in the intron preceding the terminal exon. Once again, the presence of the HERV sequence provides a site for alternative splicing, although the biological activity of the alternative protein is presently unknown [27]. These examples confirm the prediction of the AGEing model that AGEs produce specific genetic changes.

## NEW EVIDENCE THAT EXPANDS THE AGEING MODEL

Although new evidence supports the AGEing model, additional data from comparative genomics is revealing a more complex picture of the genome than previously suspected. Quite unlike the small and recognizable AGEs, large chromosomal segments can also be transposed, as several recent genome projects have revealed [36,48]. Though transposition is a mechanism associated with AGEing, chromosomal re-arrangements fall outside of the original AGE definition. Similarly, plasmids responsible for many bacterial traits are readily transferable [62]. Finally, studies have established the occurrence of regulatory elements that control AGE transposition [37,44]. One study has even shown that AGEs can be mobilized in response to microclimatic variation [29]. Because the original AGEing model does not account for these phenomena, modification (or rejection) of the AGEing model is necessary. Here I will argue that AGEing is one component of a highly regulated, genomic modularity mechanism that may still be active in modern organisms.

***Genomic Rearrangements.*** By comparing the genomes of members of the same baramin, we can detect differences that most likely arose during the historical diversification of the baramin. In other cases where precise baraminic relationship is not known, an inference of genomic rearrangement will be as reliable as our best guess as to the baraminic relationships of the organisms involved. For example, genomic differences between two strains of the same bacterial species probably arise from a historical rearrangement, but differences between bacteria and archaea were probably created. As we will see, the evidence for true genomic rearrangement is substantial, even among members of the same species.

The grass holobaramin consists of 10,000 species in some 46 different tribes, including wheat, barley, maize, rice, and oats [69]. Despite this enormous species diversity, the genomes of different grasses show many regions of similar gene order, but these regions are found in different places in different grass genomes (see Gale and Devos [23]). For example, maize chromosome 9 has conserved gene order with rice chromosomes 6 and 3, and maize chromosome 8 has conserved gene order with rice chromosomes 1 and 5. Blocks of genes like these accounted for 70% of the rice genome and 62% of the maize genome, indicating that a significant fraction of both genomes have rearranged so much that gene order is no longer conserved [1]. Similar evidence of rearrangement can be seen by comparing wheat and rice chromosomes. Wheat D Chromosome 5 has blocks of genes with the same order as four different rice chromosomes: 3, 9, 11, and two regions of 12 [22]. In each of these cases, the chromosomal rearrangements were most likely historical occurrences during the diversification of the grass holobaramin.

In another surprising example of genomic rearrangement, the sequence of *Arabidopsis thaliana* chromosome 2 contains a region that is 99% identical to the *Arabidopsis* mitochondrial genome [36].

The high degree of similarity between these sequences suggests that a copy of the mitochondrial genome was recently inserted into the nuclear genome of *Arabidopsis*. A followup study performed using fluorescent *in situ* hybridization indicates that the mitochondrial insertion in chromosome 2 is 620,000 nt [59]. As of this writing, no published analyses of other *Arabidopsis* species or ecotypes had reported a similar insertion.

In bacteria, the first evidence of chromosomal rearrangement from a genome-sequencing project came from the gastric pathogen *Helicobacter pylori*. The first *H. pylori* genome sequence to be published came from the laboratory strain 26695, and the second came from the clinical isolate J99 [2,64]. A comparison of the two genomes revealed that both strains contained unique genes not found in the other (89 in J99 and 117 in 26695). These genes are significantly concentrated in a region Alm et al. call the 'plasticity zone' [2]. In 26695, the plasticity zone is divided into two large fragments that are separated on the chromosome, but in J99, the plasticity zone is a contiguous region of the chromosome [2]. The chromosomes of these two strains exhibit non-random differences in gene content and order.

Further evidence of genomic rearrangement comes from two strains of the bacterium *Escherichia coli*. The laboratory strain MG1655 is nonpathogenic and widely used in biotechnology, but the pathogenic strain EDL933 causes haemorrhagic colitis in humans. The genomes of these two strains of *E. coli* share a total of 4.1 million nt in common, with largely conserved gene order. MG1655 contains 530,000 nt of DNA not found in EDL933, and EDL933 contains 1.34 million nt not found in MG1655. These strain-specific nucleotides are distributed in hundreds of segments throughout the chromosome. The EDL933 genome contains 1387 genes that are located in segments found only in EDL933 [48].

***Bacterial Plasmids.*** The best-known extrachromosomal features of bacterial genomes are plasmids, pieces of DNA that replicate independently of the chromosome. Plasmids can be linear or circular, and may range in size up to 1.7 million nt [19]. Plasmids and their attributes have been known for years, but with the modern emphasis on genomics, we continue to discover new examples of highly polymorphic plasmids and plasmids integrated directly into the bacterial chromosome. Although I limited my original idea of AGEs to small transposable elements (e.g. retrotransposons), plasmids are widely acknowledged to be transposons, albeit very large ones [62]. While I acknowledged the importance of plasmids as possible AGEs, I underestimated their impact on bacterial diversity. Since plasmids carry genes that confer useful phenotypes, plasmids play an important role in bacterial diversification.

Among the most economically-important plasmids are the symbiotic plasmids of the rhizobia. Rhizobia are α-proteobacteria capable of forming mutualistic symbioses with legume species. Symbiotic rhizobia can fix atmospheric nitrogen, which can be absorbed by the host plant. The genes required for mutualism are found on a plasmid in some rhizobial species but integrated into the chromosome in others. In *Rhizobium* sp. NGR234, the majority of the genes for symbiosis are carried on the 536,165-nt plasmid pNGR234a [21]. In *Sinorhizobium meliloti*, the majority of symbiotic genes occur on a plasmid pSymA, but pSymA is more than twice as large as pNGR234a and contains three copies of the nitrogen fixation genes [3]. In contrast, the symbiotic genes of *Mesorhizobium loti* are located in a 610,975-nt region of the main chromosome called the "symbiotic island." This island is bounded by a 17-nt repeated sequence, indicating that it most likely arose by an insertion of DNA into the chromosome [30]. The symbiotic genes of *Bradyrhizobium japonicum* are located on its 8.7 million nt genome, where they are similarly clustered [31].

Plasmids in other bacteria also contribute beneficial traits. *Sphingomonas* bacteria possess the ability to degrade a wide variety of cyclic aromatic hydrocarbons, including toluene, benzene, and phenol, all of which are toxic. In *Sphingomonas aromaticivorans*, aromatic hydrocarbon catabolism is associated with the plasmid pNL1. The sequence of plasmid pNL1 revealed 186 predicted genes in three distinct functional classes. The largest class, consisting of 79 genes, contained genes known to be involved in the transport and catabolism of aromatic hydrocarbons. The next class involved genes necessary for the replication of the plasmid. The final class contained several genes known to be involved in conjugation, the bacterial method of transferring DNA from one cell to another. The fact that pNL1 also contains genes allowing it to transfer itself horizontally immediately suggests that *S. aromaticivorans* acquired pNL1 by horizontal transfer; although attempts to induce conjugation between *S. aromaticivorans* and other *Sphingomonas* species failed [49].

Some plasmids have harmful effects, suggesting some kind of degradation from the created condition. For example, the toxin genes of the anthrax pathogen *Bacillus anthracis* occur on the 181,654-nt plasmid pXO1 [45]. When cured of the pXO1 plasmid, the bacteria become avirulent. Furthermore, when *B.*

*anthracis* is cured of all of its plasmids, it becomes difficult to distinguish from strains of the closely-related *Bacillus cereus* [25], a normally-benign soil bacterium that also can be an opportunistic human pathogen. Thus, in the case of *B. anthracis*, we find evidence that the acquisition of a plasmid induces an otherwise harmless bacterium to become a biological pathogen.

***AGE Transposition Control.*** The AGEing model predicts that AGEs are presently and irreversibly inert. I attributed the cessation of AGE activity to mutational degeneration. Recent research has demonstrated that AGE transposition appears to be highly regulated within the genome [44,37]. Furthermore, some AGEs retain the ability to transpose and remodel their host genome in response to environmental changes [29]. These evidences suggest that the cessation of AGE activity resulted from a higher-level control mechanism inherent in the host genomes, not because of mutational degradation.

Plant retrotransposons (AGEs that replicate via reverse transcription) are significantly methylated in plant genomes [40]. Methylation involves the addition of a methyl group to the cytosine base of a CpG dinucleotide. Researchers have observed that demethylation of plant genomes (e.g., when the methylase enzyme is mutated [26]) results in mobilization of retrotransposons [72]. Liu and Wendel reported that introgression of DNA from wild rice (*Zizania latifolia*) into cultivated rice (*Oryza sativa*) induced demethylation and activated transposition in the introgressed genome. After introgression, the retrotransposon *Tos17* showed both an increased copy number and decreased methylation. By the ninth generation of breeding, *Tos17* had ceased to transpose [37]. O'Neill et al. report similar results for marsupial hybrids. O'Neill et al. crossed the swamp wallaby (*Wallabia bicolor*) with the tammar wallaby (*Macropus eugenii*) and found an extension of the centromeres of tammar wallaby chromosomes in the hybrid offspring. This centromere extension was composed of a novel, undermethylated retroelement that was named kangaroo endogenous retroviral element 1 (KERV-1). O'Neill et al. could not detect KERV-1 in either parent species, which the authors interpreted as evidence that KERV-1 arose by recombination between two different retroelements in the parent genomes [44]. These examples illustrate a higher-level control mechanism for AGEs.

A further example of the regulation of AGE transposition comes from *BARE*-1, a retrotransposon from the barley (*Hordeum* spp.) genome. On average, 14,000 copies of *BARE*-1 exist in a given barley genome, but the copy number varies significantly and is negatively correlated with water availability. Recently, Kalendar et al. examined wild barley plants (*Hordeum spontaneum*) along a 300m transect of Evolution Canyon, Mt. Carmel, Israel. They found that the copy number of *BARE*-1 correlated both with elevation of the plant as well as the direction of the slope face. Individuals in the higher, drier sites had as much as three times the *BARE*-1 copy number as individuals in the lower, wetter sites. The variation in copy number correlated with environment further supports a higher-level control of AGE transposition.

## GENOMIC MODULARITY: EVIDENCE, DIFFICULTIES, AND FUTURE RESEARCH

The concept of transposable elements as causative agents of biological diversity is not novel, even among creationists [7,20,33,41]. I distinguished the AGEing hypothesis by emphasizing the design necessary for the successful functioning of an AGE-induced system of diversification. In order to generate the observed levels of intrabaraminic diversity, AGEs must have been designed to induce genetic change, and genes must have been designed to respond to AGEs. Without a tightly integrated AGE/gene system, AGE-induced changes would be reduced to a novel source of mutations upon which natural selection could act. As I argued previously [68], mutation and selection would be far too slow to account for intrabaraminic diversification.

My initial concept of AGEing focused exclusively on the relationship between AGEs and genes. My present survey of evidence for genomic change implies that chromosomal rearrangements are more common than specific AGE/gene interactions. These rearrangements can induce beneficial phenotypic changes only if the rearrangement is regulated and the genome is designed to respond to the rearrangments. Aberrant chromosomal changes produce well-known human syndromes (e.g. Down syndrome). I use "genomic modularity" to refer to genomic rearrangements associated with intrabaraminic diversification. AGEing is probably a minor feature of genomic modularity.

In my original presentation, I emphasized mutation as the source of modern AGEs' perceived inactivity, though I acknowledged that methylation might also play a role [68]. According to this interpretation, AGEs are presently inactive and degenerate, and therefore diversification cannot occur again. The observations discussed in this paper reveal genomic modularity that is still taking place, though presumably on a smaller scale than what must have occurred after the Flood. Present modularity activity

implies a different view of AGEing inactivity. AGEs and genomic modularity can occur in the present in response to stress. This suggests that modularity is controlled by a highly-regulated mechanism that is capable of responding to stress. In this view, modularity was activated by the environmental stress of the post-Flood world. Though presently dormant, they presumably can be reactivated by stress, possibly even inducing another episode of diversification.

The evidence of genomic modularity may be summarized as follows: First, species that belong to the same baramin are likely to have shared a common ancestor at some point in the past. Common ancestry of co-baraminic species is particularly well supported for terrestrial vertebrates, which experienced a population bottleneck at the Flood. Second, comparative genomics between co-baraminic species reveal genomic rearrangements, implying that these rearrangements took place after divergence from the common baraminic ancestor. Third, many modular genomic elements carry genes that code for important biological traits, particularly in bacteria. Fourth, genomic modularity is a highly regulated phenomenon, implying an origin by design. Fifth, genomic rearrangements occur in response to stress.

Sternberg has criticized theories of evolution caused by transposable elements because despite large genomic differences between, for example, strains of the same bacterial species, the organisms remain recognizably members of the same species [58]. Stress-induced mobilization of transposable elements has not produced a new species, despite many experiments subjecting organisms to stress. This criticism would also apply to genomic modularity and is worthy of future research. A possible solution to this problem could be found in threshold effects, that is, the level of stress placed upon organisms today may be either the wrong kind of stress or merely insufficient in magnitude to produce the diversification response. In either case, it is important to be cautious when evaluating higher-level principles (diversification by modularity) in the light of lower-level phenomena (AGE transposition). The field of ecology blossomed precisely because the first ecologists disregarded criticism from physiologists that all of ecology could be reduced to physiology [18]. In the context of genomic modularity, the diversification affects may only be apparent at higher levels of genomic organization and may not be reducible to the sum of recent TE transpositions.

Like the AGEing hypothesis, the attribution of speciation to genomic modularity is not novel to creationists. For example, Shapiro has repeatedly argued for a broader view of evolution based on 'natural genetic engineering,' a term essentially embracing the same phenomena as genomic modularity [54,55,56]. Ironically, Shapiro uses 'natural genetic engineering' as an argument against Behe's irreducible complexity [5], because mutations caused by TEs could produce coordinated, multilocus adaptations [54]. He calls his view a "21$^{st}$ Century View of Evolution," which maintains that "Major evolutionary change … occurs by the amplification and rearrangement of pre-existing modules" [55]. Though this is similar to my own view of genomic modularity, Shapiro does not explain the origin of the 'preexisting modules' or the origin of the complex cellular/genomic system capable of responding to environmental stress. Elsewhere, I argued that God would create organisms to be adaptable [70], making historical adaptation a necessary component of God's biological design. As a result, under the creationist view, the creation of both pre-existing modules of genetic information and a mechanism by which they could be rearranged would be expected.

It has not escaped my notice that the view of species transmutation outlined in this paper resembles the perspective of certain evolutionists of the nineteenth and early twentieth centuries. According to Ospovat, before reading Malthus, Darwin envisioned species transmutation as proceeding by adaptive variation. Darwin's published view in *Origin* endorsed random variation that *became* adaptation by natural selection [47, p. 43]. Though Darwin's argument for species transmutation convinced nearly all of his peers, natural selection as the causative agent of speciation remained controversial long after his death. Darwin's pre-Malthusian view of species transmutation is closer to my own view of change in response to environmental stress, although I would not claim that all species are related to a common ancestor and I would not necessarily claim that all change is adaptive. As with Shapiro, though, it is easier for the creationist to explain transmutation in response to environmental change. Creationists can attribute the ultimate source of all complex traits (adaptive or otherwise) to the design plan of God.

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