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A REVIEW OF CRS eKINDS PREDICTIVE SUCCESS AND KNOWN GENETIC MECHANISMS AFFECTING THE PREVALENCE OF ALLELES IN A POPULATION: MEIOTIC DRIVE AS A COMPETING EXPLANATION FOR PATTERNS ATTRIBUTED TO NATURAL SELECTION

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ABSTRACT

The CRS eKINDS (examination of kinds in natural diversification and speciation) research initiative has been highly successful, turning out eight full-length journal articles in less than six years. Using the history of Genesis to interpret scientific data, the project has made great strides in understanding patterns of diversity and adaptation from a biblical perspective. Two predictions were made related to how organisms adapt genetically. The first is that mutations are not random, as is commonly assumed, but rather are biased to be adaptive. This prediction was confirmed in a model plant species in 2022. The second prediction is that there are genetic factors designed to increase the prevalence of adaptive alleles. A brief overview of meiotic drive, especially biased gene conversion, is covered as a potential mechanism for accomplishing this. Taken together this implies that the commonly promoted evolutionary mechanisms of random mutation plus natural selection cannot account for the patterns we see in the world today. On the contrary, processes of diversification and adaptation require numerous, amazingly designed mechanisms, many of which we are only now beginning to understand. CRS eKINDS research highlights how the biblical narrative is useful in understanding biology and recognizing the amazing deeds of our awesome Creator.

KEYWORDS

eKINDS, biased gene conversion, meiotic drive, natural selection, predictions, adaptation, created kinds, diversification

INTRODUCTION

In 2016 the Creation Research Society (CRS) announced a bold new research initiative that was intended to catalyze biological research addressing key questions related to biblical and natural history (Anonymous 2016). Conceived by Dr. Kevin Anderson, then director of the CRS Van Andel Creation Research Center, and Dr. Jean K. Lightner, a CRS board member, this project has moved forward to become highly successful, turning out eight full-length journal articles by the beginning of 2022.

In addition to those who launched the project, other researchers have been involved. Perhaps the most noteworthy is the famed ornithologist Dr. Jon E. Ahlquist, whose early molecular biology work with Charles Sibley in the late 1970s through the 1980s led to important advances in the field of avian taxonomy (Sibley and Ahlquist, 1990). Dr. Ahlquist became a creationist later in life and was thrilled to lend his expertise to advance the creationary understanding of our world (personal communication) until he moved on to see his Maker in 2020. Also involved is Dr. Matyias Cserhati, whose bioinformatic skills have been a valuable asset.

The name of this research initiative is eKINDS, which stands for **e**xamination of **k**inds **i**n **n**atural **d**iversification and **s**peciation. This research was deemed critical because the standard evolutionary-based view of diversification is neither consistent with observational evidence nor the biblical narrative (Table 1). Diversification has been observed to occur much faster than random mutation and natural selection would logically allow. For example, in domestic species, there are hundreds and sometimes thousands of breeds or cultivars that have arisen within hundreds to thousands of years (FAO 2015; Janick and Moore 1996). Rapid speciation also appears to have occurred in many birds (Lightner 2013). Rapid diversification and speciation are consistent with the biblical timeline, but we need to understand the underlying basis of it. To aid in this process eKINDS was structured to address three basic questions.

The first question is "which organisms today are descended from the same created kind?" This is a basic question of baraminology, the study of created kinds. This field of study is derived from history presented in Genesis, where God created plant and animal life "according to its/their kind(s)" (Genesis 1:11, 12, 21, 24, 25), and uses the anglicized form of the Hebrew word for *(he) created* (bara) and *kinds* (min) to distinguish itself. This broad field is well established (Friar 2000, Wood et al. 2003, Wood 2006).

The eKINDS project was able to make a modest contribution to this field through the extensive knowledge and research done by Ahlquist on landfowl (Ahlquist and Lightner 2019). We also emphasized the importance of using multiple lines of evidence to establish what organisms constitute a probable baramin (created kind), a point that has been made elsewhere (Thompson and Wood 2018, p. 219).

Table 1. A comparison of the biblical and evolutionary views of life in natural history, logical expectations regarding design in adaptation, and the significance of these differences as they relate to scientific research.

BIBLICAL VIEW	EVOLUTIONARY VIEW
Background (history)	
A wise and loving Creator	No creator
A history – Genesis	Assumed (unobserved) history
Life was created according to their kinds – limited common descent	Universal common descent
A global flood affected life on earth	No global catastrophes are assumed
Logical Expectations	
Design and purpose should permeate our discoveries, though evidence of the Curse would also be apparent	No real purposiveness except what can arise via natural selection
Mutations are largely biased to be useful/adaptive, with evidence of errors or damage apparent as well.	Mutations are accidents/errors and are random, at least with respect to fitness.
Biased gene conversion is usually adaptive, with some examples of failure in this complex designed system.	Biased gene conversion is a "selfish" mechanism that does not help adaptation.
Other forms of transmission distortion (TRD) should be potentially adaptive under most circumstances.	TRDs mechanisms are "selfish" and "cheat." They were not designed to help adaptation.
Natural selection is not essential as design accounts for most of what evolutionists attribute to natural selection.	Natural selection is essential and is used to avoid accepting purposiveness that would imply a creator.
Significance	
The biblical worldview gives us reason to look for purpose. As we do so, the amazing design reveals God's care for his creatures, even in this fallen world. Thus, God is glorified as we more clearly see how awesome he really is.	Assuming a lack of purposiveness hinders scientific discoveries and obscures what is really going on.

Another line of research addressing the first question involved developing a method of analyzing the rapidly accumulating protein sequence data (O'Micks 2017). We used it to show that humans are profoundly unique when it comes to proteins our bodies produce (Lightner and Cserhati 2019). eKINDS has continued the development and application of this technique known as the gene content method (GCM).

Removing the misconception that all life is related by universal common descent and identifying created kinds (baramins) is only the beginning of understanding biology from a more biblical and realistic perspective. Logically following this is the second question addressed in the eKINDS research: "what mechanisms are responsible for the astounding diversity we see today within created kinds?" A further discussion of this topic will comprise the bulk of this paper.

The third question is "can we trace the natural history of animal kinds as they dispersed from the Ark to repopulate the earth?" An

initial attempt was made by Ahlquist and Lightner (2021) with landfowl. The conclusions for taxa can be heavily influenced by where one places the Flood/post-Flood boundary. It is hoped this question can be better addressed in the future.

A summary of early work was presented in a previous ICC paper (Lightner and Anderson 2018).

EVALUATING DIVERSITY WITHIN BIBLICAL HISTORY

The first step in addressing the question of the amazing diversity within created kinds is to identify the diversity that exists in groups of plants or animals where there is good evidence that they are related, and thus from a creationist standpoint, are from the same baramin. Morphological diversity in crop species was documented many years ago by a Russian scientist, Nikoli Vavilov (1922). In contrast to Darwin's belief that variation is random and unlimited, Vavilov demonstrated that clear and sometimes predictable patterns of variation exist. Some of these are very useful to humans as we use

these plants agriculturally, suggesting a Designer created the ability of plants to vary with us in mind.

More recently some of the variation within cattle (subfamily Bovinae; Lightner 2007) and sheep and goats (subfamily Caprinae; Lightner 2006) was discussed as evidence was presented that members of the respective subfamilies are related. As part of the eKINDS project, diversity in landfowl was evaluated (Ahlquist and Lightner 2020). An overall observation is that the color and morphology of various structures can often vary. For example, in bovids the size, shape, and number of horns may vary. In many taxa, the length, texture, and color of feathers or hair often vary within a kind. This phenomenon is also readily apparent in our modern dog breeds where limb and muzzle proportions vary, in addition to coat colors.

These observations further confirm that variation has non-random patterns. Variation can be adaptive for the organism, helpful to humans in the agricultural use of the species, or just provide beauty and interest in our varieties of flowers or breeds of pets. This fits well with a biblical history where God created the world. He designed plants and animals with the ability to adapt so they could fill the earth and it would be inhabited (Genesis 1; 8:16-19; Isaiah 45:18). In his kindness to us, God designed many creatures with the ability to change in ways that benefit us agriculturally. The overwhelming nature of this kindness to us is displayed in the beauty and interesting variety in Creation and our ability to appreciate and enjoy it (Psalm 104:24). The occasional appearance of gross deformity which does not fit easily in the above categories is a reminder that we live in a fallen world (Genesis 3) and should be awaiting our final redemption (Romans 8:23; Ephesians 1:13-14).

Genetic diversity also needs to be evaluated; it can take many forms. For example, there may be differences in specific nucleotide base pairs (single nucleotide polymorphisms; SNPs), apparent insertions or deletions of DNA (indels), differences in the number of copies of a specific region of DNA (copy number variant; CNV, or variable number tandem repeats; VNTR), other structural rearrangements (including inversions and chromosomal fusions/fissions), and movement of transposable elements (which are another type of indel). There has been considerable discussion of the latter as a means of inducing potentially adaptive variation (Terborg 2009), but that is beyond the scope of this paper.

A survey of genetic diversity in canids revealed that some genes in dogs have considerable variability (Lightner 2009). Genes in the major histocompatibility complex (MHC) were the most variable of the genes discussed, containing anywhere from a few dozen to over 90 different alleles. A portion of another gene had seven different alleles from VNTR. Karyotype differences were also noted in this and several other studies (Lightner 2007; Lightner 2008a; Ahlquist and Lightner 2019).

These observations then need to be interpreted within the Genesis Creation/Flood history (Table 1). While we know that only two humans were created, and all others descended from them, Scripture does not explicitly state how many individuals were created for other creatures. One might surmise that their populations were not particularly large given the command to reproduce and fill the earth (Genesis 1:22). Further, Adam named the land animals and birds, but

no suitable helper was found so God created Eve from Adam's side (Genesis 2:18-24). This may suggest that the creatures Adam named were created in pairs, with two being the most common number of creatures within a kind, to make the need for a suitable helper obvious. However, since the fall resulted in the death of animals to cover Adam and Eve with skins, there may have been more than two for some kinds, unless there was the extinction of a kind immediately following the Curse (Genesis 3:21).

At the time of the Flood, there was a significant bottleneck (drastic reduction in population size) for each kind of air-breathing terrestrial animal. The Ark included eight humans and a single pair of each kind except for clean animals and birds, which were represented in sevens. While the kinds on the Ark may not have held a one-to-one correspondence with the created kinds given it occurred significantly later in history, there is still sufficient information to be helpful to use in interpreting the information we find (Lightner 2021).

Dr. Rob Carter (2021) has considered how various starting conditions and the impact of the Flood would affect the diversity we should expect today. Clearly, the most strategic creatures to evaluate are mammals from unclean taxa. This is because they would have been represented by only two individuals on the Ark, and thus carried a maximum of four alleles (two per individual) for any specific region of DNA. Also, many mammals have been well studied and there is considerable information available on them.

Once we have evaluated the variation within a baramin and have a good understanding of the constraints the biblical history would have on the genetics of the ancestors, it is time to consider the sources of variation.

SOURCES OF VARIATION

When considering the sources of variation, it is important to consider the basics of biology that have been well established from the study of organisms in our world today. One of the basic characteristics of living things is that they respond to their environment. We have amazing examples of wonderful design in creatures that can adapt to many harsh environmental conditions including extremes in temperature, dark and nutrient-poor conditions of a cave, and the stresses of high altitudes. The initial changes always comprise physiologic and/or behavioral adaptation (Lightner 2014; Niyas et al. 2015; Rashmol et al. 2018; Tomkins et al. 2022). Therefore, in considering how animals adapt, this needs to be the starting point of our understanding. It is excellent evidence of a wise and loving Creator.

Physiologic and behavioral adaptation isn't the whole story. There are times when populations that have lived for many generations in, for example, a high-altitude environment have specific versions of genes (alleles) that have been demonstrated to be adaptive in that environment (reviewed in Lightner 2014). These are often in the same genes or pathways that are involved in physiologic adaptation to that environment.

Where do these adaptive alleles come from? There are several possibilities: created diversity, accidental changes to DNA (random mutations), and genetic changes by design (mutations that are biased to be adaptive).

In the evolutionary narrative, every allele at some point arose by

accident (random mutation). When attempting to account for adaptation, there is often a distinction made between standing variation (alleles already present in the population) and de novo mutations. If the adaptive alleles are already present, genetic adaptation can be quite rapid. If not, then it must await the appearance of a fortuitous, adaptive mutation.

Creationists recognize the need for a Creator to explain the complex genetic and biochemical structure of living things. There is plenty of room to argue for lots of created diversity. However, the Flood caused a bottleneck that would affect the diversity we see today. How can we tell the difference between created alleles and ones that have arisen via mutation (either random or biased)?

The most definitive way to identify a mutation is by doing parent-offspring comparisons. If we correctly identified the parents and the offspring, and the tests are reliable, we can have good confidence that an allele in the offspring that is not present in either parent (assuming a sexually reproducing organism) is from de novo mutation. The mechanism (accidental damage vs the result of genomic programming to generate adaptive mutations) cannot be known without considerably more study. Therefore, in this discussion mutation is simply referring to a change in the DNA sequence.

There are several other ways of inferring that mutation has occurred. For a species (or group of related species) descended from a kind preserved on the Ark, the current genetic variability can be compared to the maximum diversity that could have been present in their ancestors on the Ark. For example, dogs are unclean and some of their genes have more than four different alleles represented in the species. Thus, some of them must have arisen via mutation (Lightner 2009).

Another way of inferring an allele arose from mutation is to see how it affects the complex, biochemical pathways it is involved in. If an allele codes for a protein that disrupts a biochemical pathway, it is likely from mutation. It should be noted that many adaptive alleles disrupt a pathway; it just happens that the resulting phenotype is adaptive in a particular environment. This is even more evidence of amazing design; not only did God create amazing biochemical pathways, but their design allows for changes that are adaptive. Finally, if a new trait arises that is known to be genetic (e.g., a white horse) with no evidence it existed in the animal's ancestry, it is reasonable to suspect a mutation (Lightner 2010).

Once mutations are identified, we can look at patterns and see if they fit the standard assumption that they are from copying errors or other sources of DNA damage. The biggest problem with trying to import the standard evolutionary idea that all mutations are random errors is the evidence for rapid adaptation when new mutations are involved. Spetner (1998) examined evidence of rapid adaptation in bacteria and concluded mutations must be non-random and biased to be adaptive. Others have added to this evidence (Shapiro 2002, 2022). Based on patterns of phenotypic and genetic diversity, I have been advocating that mutations are biased to be adaptive in mammals as well (Lightner 2006, 2008b). And this was the foundation of an important eKINDS prediction.

PREDICTIVE SUCCESS

In my examination of sheep and goats (which I referred to as tsoan based on an anglicized form of the Hebrew word for flock), I considered diversity found in karyotype, horns, and pelage. I saw adaptive diversity that didn't fit well with the bottleneck of the Flood. I concluded the paper by stating:

> The variation present within the Tsoan monobaramin is from both the variety created in this baramin initially and changes that have been acquired throughout history. Some characteristics naturally change as a result of environmental changes, for example growth of a heavier winter coat and moulting. However, the variation within the monobaramin far exceeds this. Mutations, any acquired change within the genome, have historically been considered to be due to random copying errors. As such, they do not significantly add information and often result in disease. However, within the last several decades evidence has been found that some changes within bacterial genomes are directed. Such mutations can be initiated by environmental signals which allow changes in a part of the genome that is likely to help the organism adapt.[31] Much of the variation in pelage could be attributable to similar changes.[32] For example, growth in any tissue is controlled by multiple factors; some work to stimulate growth, others to inhibit growth. If directed changes occurred as a result of environmental changes from a post-Flood ice age, mutations may have occurred that increased factors stimulating hair growth and density[33,34] or decreased factors inhibiting it.[34] This would easily explain how animals which had no need for heavy coats prior to the Fall were able to acquire them when the need arose. (Lightner 2006, p. 64)

When I examined genetic diversity in the melanocortin 1 receptor (*mc1r*), a transmembrane protein involved in pigmentation in mammals, the evidence was even more astounding (Lightner 2008b). There were some SNPs that were clearly mutations that appeared in different baramins. More remarkable, there were deletions that removed nucleotides in multiples of three; this eliminated some amino acids in the middle of the protein and left the end unchanged. In most cases, it was associated with a melanistic (black) phenotype. This is because the receptor no longer responded normally to its signaling molecules. Instead, it was "stuck" in an "ON" position and always signaled for the darker (eumelanin) pigment to be produced.

One might be able to explain this improbable pattern in indels if frameshift mutations, which are not in multiples of three and would affect the other amino acids that follow it, were deleterious. However, loss-of-function mutations in this gene yield interesting variety as well, without harm. So, it appears there is bias in mutations that occur within this gene.

The pattern in humans is a little different than in other mammals. Over 60 alleles are known for the *MC1R*, which means most must have been the result of mutation because the sons of Noah and their wives could not have carried more than twelve alleles (two each). While most mutations involve some loss of function, the degree of loss varies widely. Mutations in this gene are the most common cause of red hair in humans. A few of these genes are dominant or semi-dominant and may be associated with an increased risk of melanoma.

One study (Harding et al. 2000) found this locus nearly invariant in the African populations they sampled, with just five alleles that differed at the third base pair position. This does not change the amino acid in the protein produced. These authors tried to attribute this to natural selection since darker skin is protective against melanoma. However, there are a host of reasons for rejecting this hypothesis. First, many of the known variants involving amino acid changes are recessive and/or not associated with an increased risk of cancer. So, these could not be effectively removed. Second, no variant causes cancer; it is known that other genes and environmental factors also influence whether a person develops the disease. Finally, melanoma doesn't normally develop until the end or after childbearing years. Thus natural selection is not going to effectively remove most mutations that arise. This made me suspect that environmental factors influence the rate of mutation in this gene in a way that was potentially adaptive.

Based on this previous work of looking at intrabaraminic diversity within the historical narrative presented in Genesis, I boldly posted the following hypothesis on Researchgate for our eKINDS project:

> 1) Many adaptive mutations are not the result of random genetic errors. Instead, much like mutations involved in antibody formation, there are enzymes and mechanisms (e.g., nucleotide sequence motifs) that guide the process.

Within a few years, evidence came forth that confirmed my prediction that eukaryotes have mutations biased to be adaptive (Monroe et al. 2022). The research was done with *Arabidopsis thaliana*, a plant commonly used in genetic research. The authors took pains to exclude natural selection from biasing the results. They found that mutations occur much less frequently in places where they may do damage, and much more frequently in places where they may be beneficial.

PROPAGATING ADAPTIVE ALLELES

Once an adaptive allele exists, how does it spread to be more common in a population? Traditionally, natural selection has been appealed to as the primary mechanism for the increase of adaptive alleles and the elimination of less adaptive ones. While one can always make up a good story about how this could be, there are a variety of reasons for suspecting such stories are unrealistic (Lightner 2015).

One of the best-known long-term field studies on natural selection involved the Galapagos finches (Grant and Grant 2014). They were affected by natural selection during droughts, and it removed helpful variation rather than helping the birds adapt. Thus, natural selection can work against the well-being of the population. Further, it was found that hybridization restored much of the useful variation lost from natural selection, and immigrants that remained to populate the island were not a random genetic sample of those that visited.

Creationists need to be aware of the various behavioral and ecological factors that can affect allele prevalence in a potentially adaptive way. In addition to hybridization, migration and founder effects can play a role among organisms that can choose the environment they find most suitable; bottlenecks and expansions affect allele frequency as well (Ahlquist and Lightner 2018; Lightner 2015; Lightner and Ahlquist 2017). Yet for this discussion, we will focus on genetic mechanisms that bias allele frequency, which often go by the general

name of meiotic drive.

Meiotic drive can be defined as an alteration in the process of meiosis so that in a heterozygote (individual carrying two different alleles for a gene/region) one allele is preferentially transmitted over the other. It was first described in 1928, and many examples were uncovered in the years that followed. An overview was published by Sandler and Novitake (1957) and it has remained an important topic in genetics.

Meiotic drive is sometimes referred to as a type of "intragenomic conflict" where "selfish genetic elements" bias their own transmission (Burt and Trivers 2006). Unfortunately, this emotive terminology obfuscates what is really going on. As creationists, we need to examine the data being generated in this area from a biblical viewpoint. As we do, we will have a powerful apologetic that shows the wisdom and care of our Creator in all aspects of life, including adaptation.

BIASED GENE CONVERSION

Biased gene conversion is a well-studied form of meiotic drive. During meiosis, DNA is cut by enzymes so that homologous recombination (crossing over and gene conversion) can occur. Astounding details of these highly complex, well-designed processes continue to be uncovered, and the December 2021 edition (volume 71) of *Current Opinion in Genetics & Development* was devoted to the topic of homologous recombination. For those interested in more molecular details, see the review by Sanchez et al. (2021) from that special edition.

Unlike crossing over, which swaps portions of DNA between chromosomes (though some gene conversion can accompany this), non-crossover gene conversion resolves the induced double-stranded DNA breaks by copying the sequence of one homolog over onto the other. If the copying is equally likely in both directions, then Mendelian segregation would be preserved. However, it has been found that this process tends to be biased, leading to transmission distortion. For example, it appears that breaks can preferentially occur on one chromosome, and the sequence from the unbroken homolog will be copied over onto the broken segment (Cole et al. 2012; Sun et al. 2012). Further, because a portion of the broken chromosome invades the unbroken homolog, mismatches will tend to be preferentially converted to strong (GC, which bond with three hydrogen bonds, as opposed to AT, which bond with two) nucleotides. The latter is known as GC-biased gene conversion (gBGC) and is believed to be prevalent in eukaryotic genomes (Chen et al. 2007; Glémin et al. 2015; Hämälä and Tiffin 2020; Muyle 2011).

Gene conversion can be difficult to detect directly because the tract of DNA involved is generally short. If there is no difference in sequence between the homologs in the affected region, then it cannot be detected. One study in mice looked for gene conversion in highly polymorphic hotspots. It was concluded that although the tracts are much shorter, non-crossover gene conversion was more common and widely dispersed than crossing over within the regions studied (Cole et al. 2012). It is thus predicted to have a significant influence on transmission distortion and allele fixation. This means that biased gene conversion mimics natural selection in its ability to fix alleles (Duret and Galtier 2009).

When evaluating the literature, it becomes obvious that the assumption of common ancestry has influenced conclusions on how gBCG

has affected various genomes. For example, there are unique regions in the human genome known as human accelerated regions (HARs; for a creationary review see Tomkins 2016). These are higher in GC content than their orthologs in primates, so evolutionists have inferred they have arisen via gBGC (Galtier and Duret 2007). This led to the inference that "HARs, far from contributing to human adaptation, would represent weak points of our genome, whose function needed to be preserved, in spite of the 'undesired' fixation of numerous deleterious mutations." (Galtier and Duret 2007, p. 276) This prediction isn't holding up where HARs have been investigated in detail (Tomkins 2016).

Based on the evolutionary assumption that biased gene conversion is random with respect to fitness, metanalyses and population genetic modeling have suggested that it is likely to fix deleterious alleles and thus it has been termed the "Achilles heel" of the genome (Galtier and Duret 2007), and via this "curse of the converted" significant increase of the spread of alleles associated with hereditary diseases is envisioned (Lachance and Tishkoff 2014). Interestingly, one study of human single nucleotide polymorphisms (SNPs), which used chimp data to help estimate ancestral alleles and computer predictions to "identify" harmful mutations, estimated that nearly 60% of the time gBGC works to *reduce* the spread of putatively disease-causing alleles (Necşulea et al. 2011).

This brings up two interesting points. First, where are the validated examples of human hereditary diseases that are segregating at higher frequencies because of biased gene conversion? One paper listed over 40 known genetic diseases attributable to gene conversion, but nearly all were non-allelic gene conversions, meaning that a portion of DNA was copied over onto DNA from a different, non-homologous region (Chen et al. 2007). If biased gene conversion is random with respect to fitness and as common as empirical studies suggest, there should be a wealth of examples where it is involved in spreading hereditary diseases, many of which do not even manifest until after childbearing years.

The second point is how gBGC compares to mutation. While gBGC tends to increase the transmission of strong (GC) alleles, ordinary mutation tends to produce weak (AT) alleles. So, while biased gene conversion tends to work like natural selection in decreasing variability through the fixation of alleles, when it is combined with mutation gBGC may lead to increased diversity (Boman et al. 2021). This is consistent with the fact that biased gene conversion is common in areas of high recombination and recombination is positively correlated with diversity, suggesting a creationary lens may serve better for understanding biased gene conversion.

There are multiple lines of evidence suggesting that biased gene conversion was designed by God for the benefit of his creatures. First, there are numerous enzymes that need to be expressed in a tightly controlled manner for this highly complex process to occur; this alone is a logical reason to suspect it has a purpose. Second, the significant spread of deleterious mutations, which is predicted based on the assumption that it is an undesigned, neutral process, lacks empirical support. Due to the Fall, the creation model would predict biased gene conversion can fall short of its intended good purpose, but this should be a less common outcome. This appears to be the case (Chen et al. 2007).

A third reason to suspect purpose in biased gene conversion is that there are obvious potential purposes. Diversity is considered healthy and important in populations. When combined with mutation, gBGC appears to provide a good mechanism for generating diversity. Also, biased gene conversion can help increase the spread of adaptive alleles and/or reduce the spread of maladaptive alleles, a predicted need based on a creationary review of natural selection (Lightner 2015).

Based on the above considerations, the eKINDS project now has a new hypothesis posted on Researchgate:

> Mechanisms that bias allele transmission, such as biased gene conversion and other forms of meiotic drive, will eventually be shown to contribute significantly to the propagation and fixation of adaptive alleles.

OTHER FORMS OF TRANSMISSION DISTORTION

There are many other types of meiotic drive as well as other non-Mendelian processes that can work before or after meiosis, so the term transmission ratio distortion (TRD) has been suggested to describe them in general (Camacho 2022). Recent reviews provide excellent summaries of the current state of our understanding (Arora and Dumont 2022; Clark and Akera 2021; Dawe 2022; Friocourt et al. 2023; Kruger and Mueller 2021; Pajpach et al. 2021).

Again, emotive language is often used to describe the process. Describing genes as "cheating" meiosis shows a bias that assumes Mendelian segregation should be the norm. In an evolutionary view that attempts to eschew design, this may seem reasonable. In a creationary worldview where there is a Designer who cares for His creatures, this non-random segregation provides an inviting field of study to discover its purpose as a mechanism by which God provides for and sustains his creatures today (Genesis 1; Isaiah 43:20; 45:18; Colossians 1:16-17).

TRD systems include drive elements that cause the distorted transmission in the heterozygote of a linked locus (from a small region to a whole chromosome, depending on the specifics), causing it to be transmitted in a non-Mendelian fashion. These drive elements are often found in structurally complex loci (copy number variable regions, inversions, and satellite-rich regions, including centromeres and telomers) and are enriched in regions of low recombination (Arora and Dumont 2022).

Interestingly, TRD systems are also characterized by suppressors, that can restore allele transmission to its more expected ratio. Evolutionists imagine that suppressors can magically arise via "selection pressure" and this is the basis for claiming there is an "evolutionary arms race" occurring as new drive elements and suppressors appear (Arora and Dumont 2022). One wonders how an arms race can occur without an intelligent force behind it. More likely this is from design, and as with so many other biochemical pathways, there are mechanisms to turn things on or off as needed by the organism.

One interesting TRD system is called centromere drive, which also occurs during meiosis (and, thus, can be considered meiotic drive). Centromeres connect chromosomes to spindle microtubules and enable proper segregation of chromosomes during cell division. Interestingly, centromeric DNA can be profoundly different between dif-

ferent higher-level taxa and even within a species (e.g., *Drosophila melanogaster*), and thus evolutionists consider centromeres to evolve rapidly despite their essential functions (Dudka and Lampson 2022; Henikoff et al. 2001). This is a paradox within the evolutionary worldview, as functional elements are usually believed to be conserved.

Centromere drive occurs in females, where meiosis is an asymmetric process. During female meiosis there are two cell divisions, yet only one egg is produced. The other products are called polar bodies and do not contribute to the next generation. In centromere drive, one chromosome in the heterozygote is preferentially transmitted to the egg. The best-studied experimental model systems are monkey flowers and mice (Dudka and Lampson 2022).

Centromere repeat expansions influence the recruitment of more kinetochore proteins, which assemble on the centromere and are involved in microtubule attachment. This can affect which side of the metaphase plate a chromosome ends up on. Additionally, in the mouse, the recruitment of destabilizing factors can allow the homologous chromosomes to flip which side they are on. The strength of the drive can vary depending on genetic background (Clark and Akera 2021; Dudka and Lampson 2022).

The current centromere drive hypothesis predicts that fitness costs will elicit a genome response to select for a suppressor. Fitness costs are known in monkeyflowers (reduced seed and pollen production in homozygotes for the favored chromosome), but not in mice. There has been work to identify suppressors, but there is still much to be learned about the fascinating intricacies of centromere drive (Dudka and Lampson 2022).

While TRD in females often exploits the asymmetry of meiosis, TRD in males tends to be the result of post-meiotic mechanisms. The best-known examples are segregation distorter in *Drosophila* and t-driver in mice. They operate while sperm cells are connected by syncytial bridges and some gene products are shared. The TRD mechanisms are often classified as either target-killer or poison antidote drive systems, depending on the specifics. The driven alleles can significantly affect fertility in male heterozygotes, and in some cases are lethal in the homozygote. Interestingly, however, mice carrying the t-haplotype seem more prone to migrate and female carriers have a lower activity level and longer lifespan (Arora and Dumont 2022; Kruger and Mueller 2021).

Yeast also undergo symmetric meiosis, and similar TRD mechanisms have been identified in ascomycetes (Lohmar et al. 2022; Nuckolls et al. 2022, Zanders and Johannesson 2021). The details of these various forms of TRD are numerous and nuanced. At this point it is evident that we have much yet to learn so we can properly interpret what is going on. While some examples include adverse outcomes (low fertility; lethal in homozygotes), others do not. We need to be looking for design and purpose as we investigate to better understand what is going on.

CONCLUSION

The biblical history provides a robust foundation for understanding the world around us. The CRS eKINDS project has used the history presented in the Bible, molecular data, and scientific literature to better understand the biological realm of our world. Not only are many observations easier to explain within a biblical worldview,

but it has allowed for testable predictions to be made. One of the eKINDS predictions was that mutations (changes in the DNA sequence) are not random with respect to fitness in eukaryotes; this prediction was recently confirmed in a detailed genetic investigation of the plant *Arabidopsis thaliana*. A second prediction is that mechanisms that bias allele transmission, and thus mimic patterns expected from natural selection, will be shown to be important in fixing adaptive mutations.

The implications are that the naturalistic mechanisms we have been taught in biology (random mutation and natural selection) cannot explain what we observe. Designed mechanisms are essential for adaptive mutations to appear, and designed mechanisms are necessary for them to spread in the population in a timely fashion so God's purpose of the earth being inhabited can be realized (Isaiah 45:18). This means everything about useful genetic changes that advance adaptation and agriculture point to our Wise Creator who designed life in a way so that it could reproduce and fill the earth (Genesis 1).

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