Developmental system plasticity—a brief initial assessment of extent, design, and purpose within the creation model

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It seems reasonable to expect that pathways underlying essential characters would remain largely static. However, scientific evidence clearly shows this is not always the case. Evolutionists, having a naturalistic model, have no reason to believe that changes in developmental systems are designed in any way. To my knowledge, no creationist has ever specifically predicted plasticity in developmental pathways based on it being a logical necessity in creatures designed to reproduce and fill the earth. However, it is clear from the scientific literature that such developmental system plasticity does exist, and sometimes it is a basis for speciation within created kinds. Given the problems that are observed in hybrids, the parental species of which have undergone developmental system shift, it seems unreasonable to assume that the underlying mechanisms are purely naturalistic (i.e. random mutation and natural selection). Rather, the initial assessment presented here suggests that creatures were designed with genomic plasticity as a means to be able to adapt as they reproduced and filled the earth. There is reason to believe that future research will further uncover mechanisms indicating that there is cellular and/or genomic control of this process.

Homology has long been used as an argument to support universal common ancestry. Similar structures which are believed by evolutionists to have been inherited from a common ancestor are known as homologous. Creationists have pointed out that common design used by a Creator is an equally plausible explanation for these similar structures. For example, a pentadactyl limb is found in most tetrapods and evolutionists proffer it as evidence of common ancestry.[1] Creationists have countered that scientists have uncovered very different developmental pathways by which they are formed. This is put forth as evidence that these structures are not from common ancestry, but more likely from a common design used in different created kinds.[2]

While the creationist argument seems plausible enough, it might lull us into thinking we have a magic bullet to identify when creatures are from different created kinds. Is it reasonable for us to assume that differences in underlying developmental systems are always clear evidence that two creatures are from separate kinds? Is developmental plasticity something that would be ruled out in a creationary paradigm? Emerging scientific discoveries regarding plasticity in developmental networks have surprised evolutionists. Creationists need to understand these findings in the context of a biblical model.

Developmental system drift

It seems reasonable to assume that pathways underlying homologous characters would remain largely static. Homology in physical structures would be expected to reflect homology in the underlying molecular mechanisms producing those structures in creatures that are actually related. However, scientists have been confronted with numerous studies that indicate developmental pathways do indeed diverge over time. This can take place without any apparent change in phenotype. When this occurs, the phenomenon has been called developmental system drift (DSD). DSD is distinguished from genetic drift, but both use the term 'drift' because chance, not selection, is believed to be the underlying mechanism.[3]

When evaluating conclusions in the scientific literature, it is important to distinguish between experimental results and model-based inferences. In some cases DSD is a conclusion based on the assumption of universal common ancestry. In other words, divergent taxa, which creationists would recognize as belonging to different kinds, are being compared. In these cases creationists would not accept this as evidence for DSD because these different kinds were separately created and there is no reason to assume the underlying developmental systems were ever identical. However, there is considerable evidence for this phenomenon occurring within created kinds. Understanding the basis and extent of DSD is essential for advancing the creation model and maintaining sound apologetic arguments.

Step 1—identify created kinds

To evaluate DSD within the creation model we must be able to recognize when two or more species belong to the same created kind There are several methods that can

be used, but the most widely accepted is the ability of the two species to form hybrid offspring.[4] This is based on the understanding that God created creatures according to their kinds to reproduce and fill the earth (Genesis 1:20–25). From this it is commonly inferred that these creatures reproduce according to their kinds.[5]

This inference seems strong given that reproduction is a complex process which requires an amazing amount of coordinated expression between maternal and paternal genes. Sometimes this breaks down. There are examples where hybrid offspring are infertile, such as in the mule. Other times they rarely, if ever, get past early stages of development. For example, sheep and goats can mate, but normally they don't produce live offspring. There are a few notable exceptions where a live hybrid was produced, which is the basis for recognizing sheep and goats as being from the same kind, despite being classified as different genera.[6]

How much development is necessary in hybrid offspring before we can recognize their parents as from the same created kind? Certainly, more than just fertilization is required since human sperm can fertilize hamster eggs in the laboratory. Even the first few cell divisions are under maternal control. So, it is recognized that there needs to be significant coordinated expression of paternal and maternal genes in the embryo. It has previously been suggested that development to an advanced blastocyst should qualify.[7] Also, if two species have both been shown to hybridize with a third species, all three are considered to belong to the same created kind.

From the literature, examples were selected that would qualify as within kind comparisons according to the above criteria. Examples considered here come from the genus *Drosophila* (fruit flies where hybrids commonly reach adulthood), *Caenorhabditis*, (where the two species examined have hybridized with a third species in that genus and some hybrids in each cross have developed past the blastula stage through gastrulation[8]), and *Mus musculus* (the house mouse).

Step 2—examine evidence within a created kind; look for patterns

Fruit flies and DSD

True and Haag[9] have summarized several examples of DSD which have been identified within the genus *Drosophila* (figure 1). Both *D. melanogaster* and *D. simulans* have a conserved pattern of thoracic bristles in the adult fly. However, hybrids between these two species often exhibit a lack of bristles. This pattern of bristle loss is not seen in hybrids between *D. simulans* and *D. mauritiana*, which are believed to be more closely related. An X-linked locus has been identified which accounted for over half the variation. This locus showed significant epistatic interactions with autosomal loci.

A second example is illustrated in hybrids of *D. subobscura* and *D. madierensis*. Many exhibit aberrations in the development of the second (T2) and third (T3) thoracic segments such that they appear more like the first (T1). This can be recognized by the ectopic expression of the male-specific sex comb. The sex comb is a bristle pattern found in the front legs (on T1) of male *Drosophila* species. In these hybrids, the sex comb is present on the T2 and T3 legs as well. Genetic analysis indicates at least five loci contribute to the loss of sex comb suppression in T2 and T3. Maternal effects may play a role as well.

A third example involves regulatory divergence in *Drosophila*. The 'stripe 2' enhancer region of the *evenskipped* (*eve*) gene is a 480 bp sequence in *D. melanogaster*. It regulates expression of the gene within the second of seven transverse stripes in the embryonic blastoderm. Twelve transcription factor binding sites were identified within the enhancer in this species: six for activators and six for repressors. When the *eve stripe 2* enhancer region was sequenced in five other *Drosophila* species, it was found that they varied in DNA sequence, spacing between the binding sites, number of binding sites, and total length. Surprisingly, the enhancers from other species were able to drive *D. melanogaster* in an apparently normal spatiotemporal pattern.

So could it be that the sequence differences in the *eve stripe 2* enhancer region are not functionally relevant? To test this hypothesis, Ludwig and colleagues[10] constructed chimeric enhancers, with one segment of the enhancer derived from *D. melanogaster* and the other from *D. pseudoobscura*. The chimeric enhancer did not drive gene expression in the wild-type pattern. Thus, while the first two examples appear to involve mutations at separate loci in the genome, this latter example suggests compensatory mutations occurred within the enhancer region itself to maintain the conserved expression pattern.

Nematodes and evaluating the extent and significance of DSD

Caenorhabditis elegans (figure 2) and *C. briggsae* are nematodes which occupy the same niche and appear strikingly similar. They are believed to be closely related. Up to the 350-cell stage of embryogenesis, the lineages and timing of cell divisions are virtually identical.[11]

Despite the phenotypic similarities, their genomes have diverged significantly with only ~60% of their genes having 1:1 orthologues. They have many species-specific expansions, losses, and chromosomal rearrangements. Thus, it seems clear that while the biology of the worms has been conserved, the underlying molecular pathways have diverged significantly.

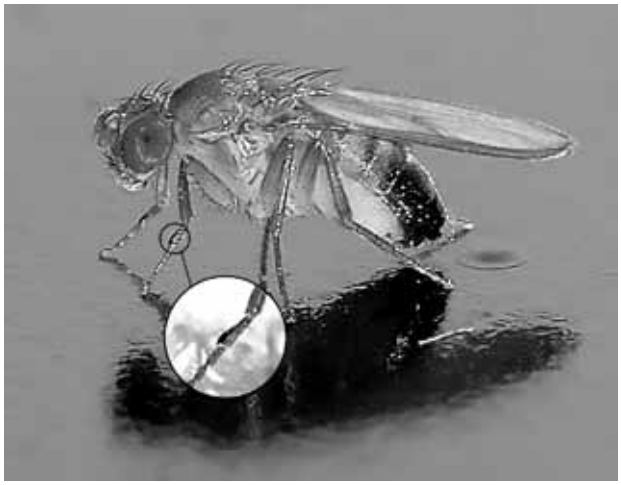

Figure 1. The common fruit fly (*Drosophila melanogaster*) has been used extensively for genetic research. Note the bristles on the thorax and the sex comb (dark region on the front pair of legs) in this male. While these features are found in other species of *Drosophila,* some hybrids lack the normal pattern of these traits.

Verster and colleagues[12] set out to examine in a systematic manner how DSD affects genes by comparing these worms. They wanted to find examples of orthologous genes playing a different role in each of these species. They reasoned that different loss-of-function phenotypes would indicate a change in function of the gene. Using RNA interference (RNAi) they screened for differences in the knockdown phenotype in more than 1,300 orthologues. The results are intriguing.

Based on their study, they estimate about 25% of the orthologues differ in their function between *C. elegans* and *C. briggsae*. Transcription factors and taxonomically restricted genes (TRGs, also known as lineage-specific genes or orphan genes) are overrepresented in this group. Protein coding genes are underrepresented. Some of this did not surprise them; they reasoned that the basic machinery of the eukaryotic cell was preserved, and transcription factors are generally seen as evolving more rapidly in the evolutionary model. However, they never suspected that TRGs would be so commonly involved. In the evolutionary model, these are recently evolved genes which now seem to be evolving surprisingly fast.

Verster and colleagues then attempted to elucidate the underlying molecular mechanisms involved in the shift in function; three were examined. First, in some genes changes in expression were involved; one of these cases was investigated to the extent of confirming that promoter evolution resulted in functional divergence. A second mechanism appears to be related to protein coding changes. In the few cases they tested they were not able to show conclusively any specific cases where differences in RNAi phenotype were the result of coding sequence changes; however, they did note a trend that the more divergent the protein sequence, the more likely that the knockdown phenotype would be different. Finally, they also found some examples where the difference in RNAi phenotype was attributable to changes in other genes.

One example they found of the latter mechanism is intriguing. Two genes, *bli-4* and *bli-5*, act together and are involved in molting, but the knockdown phenotype for them differs between the two species. Verster and colleagues hypothesized that instead of independent functional changes in these genes, there were changes in the requirement for the entire pathway due to changes in other genes. This was demonstrated by using transgenic rescue experiments which showed that the coding sequences and promoter region together were functionally interchangeable. In other words, for example, the *bli-5* gene from *C. briggsae*, under control of the *C. briggsae* promoter, was able to rescue a *C. elegans* null mutant for the *bli-5* gene. The same is true of the *bli-4* gene. The authors did a subsequent brief screening which suggested there are other examples where this phenomenon is occurring, including the genes *rsp-3* and *rsp-6*.

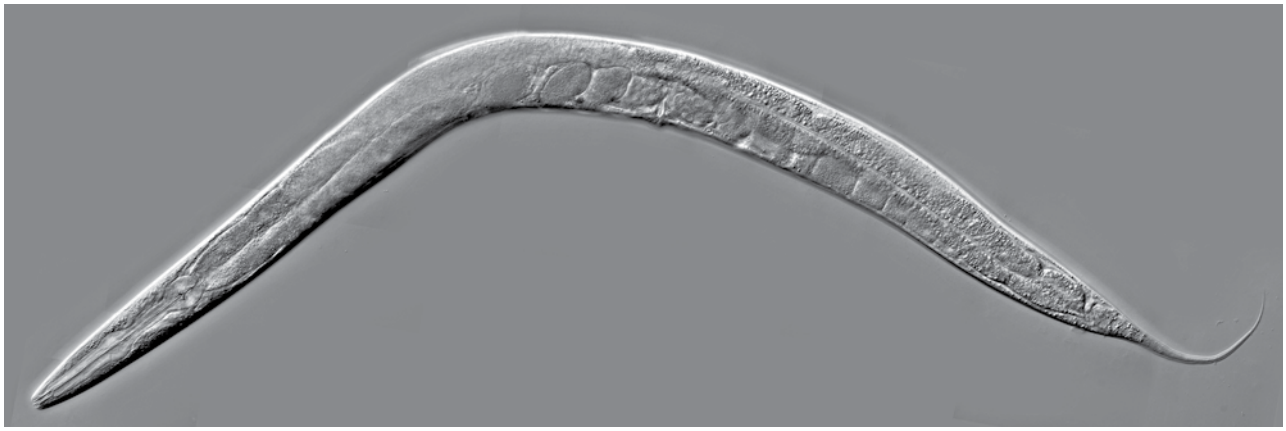

Figure 2. The nematode *Caenorhabditis elegans* has been extensively studied and the developmental fate of each cell has been mapped.

This potentially has important implications. It is common for there to be multiple redundancies among members of a gene family. Verster and colleagues suggest that this allows for the requirement for any single member to be fluid over time. They further point out that three mechanisms—changes in gene expression, rapid changes in TRGs (i.e. orphan genes), and subfunctionalization among related gene family members—are considered central molecular drivers for adaptation. Thus, they propose the possibility that DSD, where there is no obvious change in phenotype, and adaptation are somehow related.

Role in speciation as seen in the house mouse

Hybrid sterility and inviability are associated with speciation. It is recognized that negative epistasis between mutations at interacting genes is commonly the basis for this. This extends beyond the examples already described previously in invertebrates. The house mouse (*Mus musculus*) (figure 3) is a particularly useful model organism to understand the early phases of speciation; many recent studies have uncovered important details in this regard.

There are two subspecies of house mouse which play a prominent role in these studies: *M. m. musculus* and *M. m. domesticus*. Sterility in hybrids is asymmetric. Male hybrids with *musculus* mothers are nearly always sterile, while those with *domesticus* mothers are fertile. Female hybrids are fertile; a pattern consistent with Haldane's Rule. Briefly, this 'rule' describes the pattern that when one gender is absent, rare, or sterile among hybrids, it will be the heterogametic sex. The heterogametic sex is male in mammals (XY) and Drosophila (XO), but female in birds and Lepidoptera (ZW).

Various studies involving the house mouse have identified an important role for the X chromosome and several interacting autosomal loci, including *Prdm9* on chromosome 17.[13] In sterile males incompatibility between the *Prdm9* and an interacting gene on the X chromosome (*Hstx2*) is associated with impaired heterosubspecific chromosome synapsis and meiotic arrest. In other words, the incompatibilities interfere with chromosome pairing when the two chromosomes are each from a different subspecies; the result is failure to complete meiosis. Autosomal loci on chromosomes 3, 9, and 13 can alter the asymmetric sterility; certain variants are associated with male sterility regardless of the direction of the cross.[14]

Expression studies involving both F1 and F2 hybrids have revealed that sterility is associated with major alterations in genome-wide expression. Some sterility loci are involved in multiple incompatibilities; this violates a common assumption in theoretical models that incompatibilities act independently. Turner and colleagues noted in their research that changes in transcriptional regulation, which are recognized as important in adaptation, also appear to be important in reproductive isolation.[15]

Step 3—identify the questions that arise

The amount of genetic change that this suggests has occurred may be surprising for those unfamiliar with previous 'within kind' comparisons that have been published in the creation literature.[16] The pattern here brings up new questions as well as old. How can the same phenotype be maintained when there are so many underlying genetic changes that have occurred? How was viability maintained while these changes were taking place, especially considering that some of these combinations are lethal in the embryo? What are the underlying mechanisms that cause the genetic changes, and how do specific alleles become characteristic in a species? Before we attempt to answer these, it would be helpful to look at how evolutionists have viewed speciation.

Dobzhansky-Muller model of speciation

A paradox existed when Darwin proposed that natural selection was the main mechanism of evolution. How could hybrid sterility routinely evolve? Natural selection should eliminate it! A resolution of this paradox was proposed decades later in what is now known as the Dobzhansky–Muller model of speciation. The idea is that hybrid infertility is the result of two or more interacting genes. The first mutation that arises is compatible with the original genetic background. Perhaps it gets fixed in one population due to some advantage it confers. Then a different mutation arises in a different population. Again, it is compatible on the genetic background on which it appears, but proves to be maladaptive when combined with the first mutation. In this way new alleles can arise without passing through an adaptive valley.[17] This emphasizes a well-known phenomenon: the effect of a mutation is often dependent on the genetic background on which it is found.

Figure 3. The house mouse *(Mus musculus)* is a familiar animal that is commonly used in genetic and medical research. Two of the subspecies are in the early phases of speciation.

How does one conveniently get mutations to appear on the right background in an evolutionary model? Essentially, underlying mutations are believed to be random, and natural selection eliminates them when they appear on the wrong background. Infertility would certainly prevent passing on such mutations. However, only one sex is infertile in many cases; this allows the possibility of passing them on at a reduced frequency. More difficult to explain in the evolutionary model is the way so many mutations accumulate that are compatible on their background and incompatible with other backgrounds. In fact, how does one propose the origin of developmental systems that even tolerate this type of change? A naturalistic origin of such a system seems absurd, as extensive foresight and intelligence is the only known mechanism by which such elegant complexity has ever been observed to arise.

Even for the changes that occur within developmental systems designed with such astounding plasticity, random mutation and natural selection are not likely to be major mechanisms of this change. In this case, why is it so common for new alleles to arise and be fixed which simply maintain the same phenotype? Further, natural selection may account for the elimination of alleles when they have strong negative interactions—such as infertility, but it is not a viable mechanism for eliminating most deleterious mutations since their effects are generally less dramatic. Random mutation within the genome should generate deleterious mutations at a much higher rate than beneficial or neutral ones. Given realistic parameters, it has been shown that natural selection cannot stop the fixation of massive numbers of deleterious mutations by genetic drift relative to the number of beneficial mutations that can become fixed in that time.[18] This reality suggests that natural selection cannot account for the coordinated changes that have occurred within developmental systems.

Step 4—consider the evidence within a biblical model

In a biblical worldview it is recognized that God created creatures according to their kinds and blessed them to reproduce and fill the earth (Genesis 1:20–22; 8:15–19). The adaptation which occurs in so doing is expected to point to the Provider who cares for his creation (Psalm 147:7). Thus, genetic changes in populations, especially of animals like vertebrates, should not be expected to arise by the naturalistic methods of chance mutations and natural selection. In fact, several other probable mechanisms for such changes have been reviewed in the creation literature.[19] This suggests to me that the term 'drift' is probably not accurate; instead we are observing developmental system shift.

When I first started reading on DSD, I did wonder if it was somehow related to adaptation. I reasoned that it could modify the genetic background and allow adaptive mutations to arise that might not be adaptive on a different genetic background. Thus, I was encouraged when I found two research papers where the authors had proposed that DSD and adaptation may somehow be related. Given the potential for genome-wide dysregulation, which is seen in some hybrids, it seems hardly plausible to me that these changes really have 'chance' as their underlying basis. Rather, only God could have designed things so this process could occur while maintaining viability and reproductive ability.

The suggestion that initial mutations are generally compatible with the genetic background on which they appear is far more reasonable in a creation model where a Designer may have programmed the genome to operate in this way. However, at times an adaptive mutation has been observed to have fitness costs, and a compensatory mutation arises that relieves it. An example of the latter is found in sheep blowflies (*Lucilia cuprina*), where the allele conferring resistance to diazinon was associated with asymmetry and fitness costs; a second mutation at a different location is believed to have relieved this effect.[20] Either way it is suspiciously convenient that such options exist for adaptation, pointing to a caring Provider.

What about the significance of orphan genes? In many cases TRGs are associated with essential functions, meaning that loss of them is lethal.[21] Evolutionists were surprised by how widespread these genes are, and believe they recently evolved.[22] Paul Nelson of the Discovery Institute considers this implausible and discusses them as evidence against Neo-Darwinism.[23] They have also been discussed in the creation literature, with the suggestion that perhaps they could be used to identify the boundaries of created kinds.[24] However, it is important to recognize that TRGs can have a sporadic distribution within groups that are inferred to be from the same kind, based on both profound phenotypic similarity and hybridization. This is certainly the case in *Drosophila*.[25]

Since the creation model recognizes a Creator who provides, it is possible that TRGs arose from non-coding DNA via non-naturalistic (i.e. not neo-Darwinian, but designed) mechanisms. This would be indicative of impressive programming within the genome that controls genomic changes. Alternatively, they may have been created as auxiliary genes that could carry the load of developmental and/or physiologic function as developmental system shift takes place. In this case they may have been recently lost, at least in protein-coding function, rather than recently gained by a sister group. The creation model does not predict the specific initial condition, and so further investigation is warranted.

Finally, the underlying shift that has occurred in developmental systems appears to provide an explanation for why speciation occurs. Speciation is not just a matter of deleterious mutations arising, but involves underlying shifts where compatible mutations arise that allow interfertility to be maintained within each species. It is only after significant divergence that crossing parent species results in problems in hybrid offspring.

Summary

It seems clear that God created the genome with considerable plasticity. Evolutionists, who have been conditioned to think of all change as evidence of universal common ancestry, may feel this genomic change supports their view, but the reality is that it does not. The fact that changes must be made in a way to maintain viability and fertility within the species militates against such a view. Future research should uncover mechanisms indicating such changes are under cellular or genomic control. Thus, when better understood, this phenomenon will point all the more clearly to the biblical Creator who created and sustains life on Earth, despite the current fallen condition.

Glossary

Allele—one of several forms of a particular gene.

Autosomal—referring to chromosomes that are not sex chromosomes.

Ectopic—occurring in an abnormal place or position.

Epistasis—a condition where the effect of one gene depends on the allele at one or more other loci.

Epistatic—the adjectival form of the word is 'epistasis'.

Heterosubspecific—from different subspecies.

Homology—similarities which are believed to exist due to common ancestry.

Loci (singular: locus)—the specific locations of the genes.

Maternal effects—a situation where the phenotype of the offspring is influenced by the environment and/or genotype of the mother.

Orthologue—one of two (or more) homologous gene sequences found in different species.

Pentadactyl—literally having five digits (fingers/toes); it can also apply to a pattern of bones in a limb with one bone extending from the shoulder (humerus); two below it (radius and ulna) followed by wrist bones and digits.

Subfunctionalization—when each member of a duplicated gene diverges by retaining a different part of the original function of the gene. In many cases the duplication is inferred rather than observed, as with members of a gene family.

Tetrapod—an animal having four limbs, e.g. amphibians, reptiles, birds, and mammals.

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