Neutral Model, genetic drift and the Third Way—a synopsis of the self-inflicted demise of the evolutionary paradigm

Jeffrey P. Tomkins and Jerry Bergman

Because of grievous deficiencies in the standard neo-Darwinian Model of evolution, which is largely selection driven, scientists proposed an alternative postulate called the 'Neutral Model' in the late 1960s. The Neutral Model is also mutation driven, but selection is deemed to be an insignificant force of change. Instead, random genetic drift is alleged to be the main driver. Since its inception, the Neutral Model has come to be incorporated in many theoretical evolutionary scenarios at some level. However, due to numerous discoveries in genomics and genome function, the Neutral Model has also become deficient, prompting a new move in science called the 'Extended Evolutionary Synthesis' or 'The Third Way', which takes a position of blissful ignorance and offers nothing tangible to extend or support evolutionary theory. While Third Way proponents recognize the deficiency of all popular evolutionary models, they maintain that more research is needed to elucidate unknown evolutionary mechanisms and processes despite the fact that the progress of scientific discovery is revealing nothing but unimaginable complexity.

Neutral Model evolutionary theory is considered by its supporters to be the primary mechanism underlying macroevolution and, for all practical purposes, has been integrated at some level into most modern evolutionary models. The Neutral Model is also a key component in the human evolution paradigm and plays a direct role in questions concerning 'junk DNA' in the genome.

As opposed to the standard neo-Darwinian Model (also known as the modern synthesis which is largely selection driven, the Neutral Model proposes that, at the molecular level, mutation-driven evolutionary changes are not primarily acted upon by selection, but are subject to random genetic drift.[1–3] Neutral Model theorists do not completely discount selection as a factor in evolution, but limit its theoretical impact to varying levels, depending on the proponent(s) and specific scenario being postulated. The overall theory proposes that when environmental conditions change, random mutational changes that result from genetic drift may have produced a new gene or altered the regulatory control of a network of genes that turn out to be beneficial.

Motoo Kimura, one of the original pioneers of the Neutral Model, in his seminal book *The Neutral Theory of Molecular Evolution*, defines it as follows:

> "The neutral theory holds that at the molecular level most evolutionary change and most of the variability within species are not caused by Darwinian selection but by random genetic drift of mutant alleles that are selectively neutral or nearly neutral. The essential part of the neutral theory is not so much that molecular mutants are selectively neutral in the strict sense as that their fate is largely determined by random drift."[4]

Both neo-Darwinism and the Neutral Model are ultimately mutation driven for the production of molecular variation as fodder for evolutionary processes to act on. The key principle in the Neutral Model for this variation to allegedly promote evolution is that of stochastic, or chance, processes. Kimura proposed that "the great majority of evolutionary mutant substitutions at the molecular level are caused by random fixation, through sampling drift, of selectively neutral (i.e. selectively equivalent) mutants". Kimura goes on to say that this "is in sharp contrast to the traditional neo-Darwinian (i.e. the synthetic) theory of evolution, which claims that the spreading of mutants within the species in the course of evolution can occur only with the help of positive natural selection".[5]

Since its inception, Neutral Theory has earned the support of many leading evolutionary researchers. As the late Harvard Professor Steven Jay Gould wrote in 1989, Neutral Theory "has been challenging conventional Darwinism with marked success during the past twenty years".[6] Kimura, then at the National Institute of Genetics in Japan, wrote that he proposed Neutral Theory because many molecular research findings were "quite incompatible with the expectations of neo-Darwinism".[4]

The Neutral Theory was largely devised by Kimura as a resolution to Haldane's dilemma, which seriously challenged neo-Darwinism. Kimura himself stated, "the calculation of the cost based on Haldane's formula shows that if new alleles produced by nucleotide replacement are substituted in a population at the rate of one substitution every 2 yr, then

the substitutional load becomes so large that no mammalian species could tolerate it".[7] His answer to this quandary was that "the very high rate of nucleotide substitution which I have calculated can only be reconciled with the limit set by the substitutional load by assuming that most mutations produced by nucleotide replacement are almost neutral in natural selection".[7]

Other notable scientists who were devising comparable models during this era, such as Jack King and Thomas Jukes, were concerned that Kimura's estimates for genomic substitution rates were probably exaggerated.[8,9] In more recent history, theoretical geneticists have put forth a variety of models that incorporate different levels of neutrality and selection in regard to evolution.[1,2,4,10]

Random genetic drift

The concept of random genetic drift plays a pivotal role in the Neutral Model. Neutral theorists argue that "there is agreement that both random drift and selection are important in evolution; there is disagreement, however, on the relative contribution of each force".[11] Genetic drift can be described by changes in the frequency of a gene or other DNA sequence variant in a population that by itself does not confer to the organism a natural selection advantage (generally defined in terms of reproductive success). Such mutational variants are assumed to be neutral or near-neutral in their effect on the genome because they do not affect reproduction to any measurable degree. Another factor is that the alleles existing in the organism's offspring are only a sample of those existing in the parents; thus, chance plays the key role in determining if a given individual has a specific allele. A population's allele frequency results from a fraction of the copies that survive in each generation. Neutral Theory supporters postulate that because detrimental variants are rapidly purged by natural selection, they do not make significant contributions to the variation within and between species at the molecular level. Conversely, genetic drift causes most genetic variants to disappear completely. Using numerical simulation and evolutionarily favourable parameters with the Neutral Model, Rupe and Sanford showed that the vast majority of neutral mutant alleles fail to become fixed, with the problem becoming more pronounced as population size increases.[12]

The chance events that produce a zygote in a population have been compared to the random draws of marbles from a jar. Assuming there were four different alleles of a gene, they would not be selected in their exact predicted ratios of 0.25, but in ratios that may, by chance, have a few more or a few less (figure 1). Over succeeding generations, these ratios would become more and more skewed. Herron and Freeman provide the following easily understood example:

> " ... random discrepancy between theoretical expectations and actual results is called sampling error. Sampling error in the production of zygotes from a gene pool is genetic drift. Because it is nothing more than cumulative effect of random events, genetic drift cannot produce adaptation. But it can … cause allele frequencies to change. Blind luck is, by itself, a mechanism of evolution."[13]

But are genes accurately represented as marbles in a jar? The problem is that observations of declining diversity for species within real ecological systems do not support this concept. The well-known evolutionist William Provine, in his book *The 'Random Genetic Drift' Fallacy,* systematically describes that what scientists have called genetic drift is actually the effects of inbreeding. Provine documents that all the key experiments performed between 1940 and 1957 allegedly documenting the idea of drift pointed to nothing more than the consequences of inbreeding.

Of course, inbreeding leads to very different genetic outcomes than do hopeful evolutionary speculations about drift. In reality, the concept of a gene pool is an antiquated

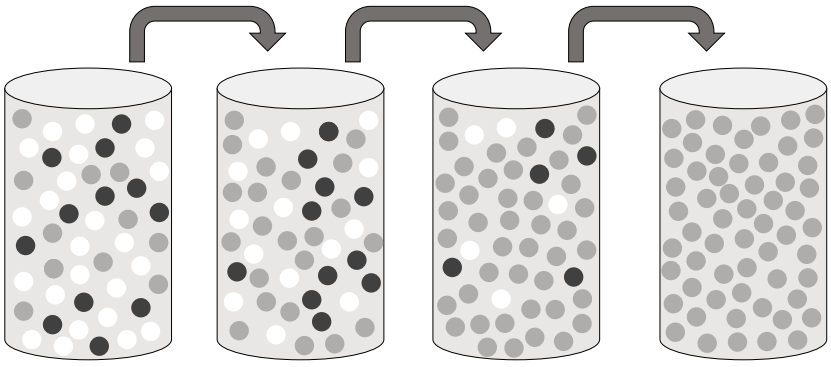

Figure 1. The hypothetical model of genetic drift can be illustrated using *N* number of marbles in a jar to represent *N* organisms in a population. Consider the jar on the far left as the starting population. The different patterns of marbles in the jar correspond to different alleles of a gene in the population. In each successive generation, the organisms (marbles) randomly reproduce. Creating the next generation can be simulated by randomly selecting a subset of marbles from the original jar and depositing it in a new jar. The second jar likely contains marble ratios different than the first jar, such that a random shift has occurred in gene allele frequencies. This process can be repeated a number of times, randomly reproducing each generation of marbles to form the next. The fluctuation of alleles is analogous to genetic drift—a change in the population's allele frequency resulting from a random variation in the distribution of alleles from one generation to the next. It may be that, for some reason, only a certain type of organism produces offspring after this process has gone on for several generations in a small population. In this case, fixation can occur as depicted in the last jar.

model devised well before discoveries about chromosome architecture became available in the genomics revolution. Genes cannot be defined anymore as simple heritable units because not only are they large and complex, but physically linked to other genes and regulatory features in genomic neighborhoods and networks of control (figure 2). Furthermore, the cellular system of genetic recombination is a highly controlled process involving both hotspots (recombination sites) and protected areas where recombination is not allowed.[14,15]

Noted evolutionist and Neutral Model drift proponent Michael Lynch actually takes this level of genomic complexity into consideration. For the record, Lynch candidly acknowledges the lack of explanatory power in the neo-Darwinian modern synthesis for explaining the evolution of gene networks, stating, "Although numerous investigators assume that the global features of genetic networks are moulded by natural selection, there has been no formal demonstration of the adaptive origin of any genetic network", and, "the mechanisms by which genetic networks become established evolutionarily are far from clear".[16]

So, what sort of solution does Lynch put forward to explain the evolution of complex gene networks? Amazingly, he proposes a completely speculative Neutral Model solution on a grand scale where complex interlocking gene networks 'magically' evolve through random genetic drift. Lynch states, "many of the qualitative features of known transcriptional networks can arise readily through the non-adaptive processes of genetic drift, mutation and recombination, raising questions about whether natural selection is necessary or even sufficient for the origin of many aspects of gene-network topologies".[16] While Lynch comes to grips with the inadequacy of the neo-Darwinian paradigm, his Neutral Model speculation, devoid of any real molecular mechanism that can create new sets of interconnected genes, is clearly even more fanciful and improbable. Networked genes actually place severe functional constraints on gene evolution. A mutation in one gene or regulatory element will affect all other connected genomic regions.

Junk or function

A major difficulty with Neutral Theory is the assumption that most DNA is non-functional. The idea of codons (an idea which included the discovery that the third base of many amino acid-specifying codons can be variable) had been elucidated less than a decade prior to the late 1960s advent of Neutral Theory. At this time Kimura and others immediately jumped on this discovery of codon variability

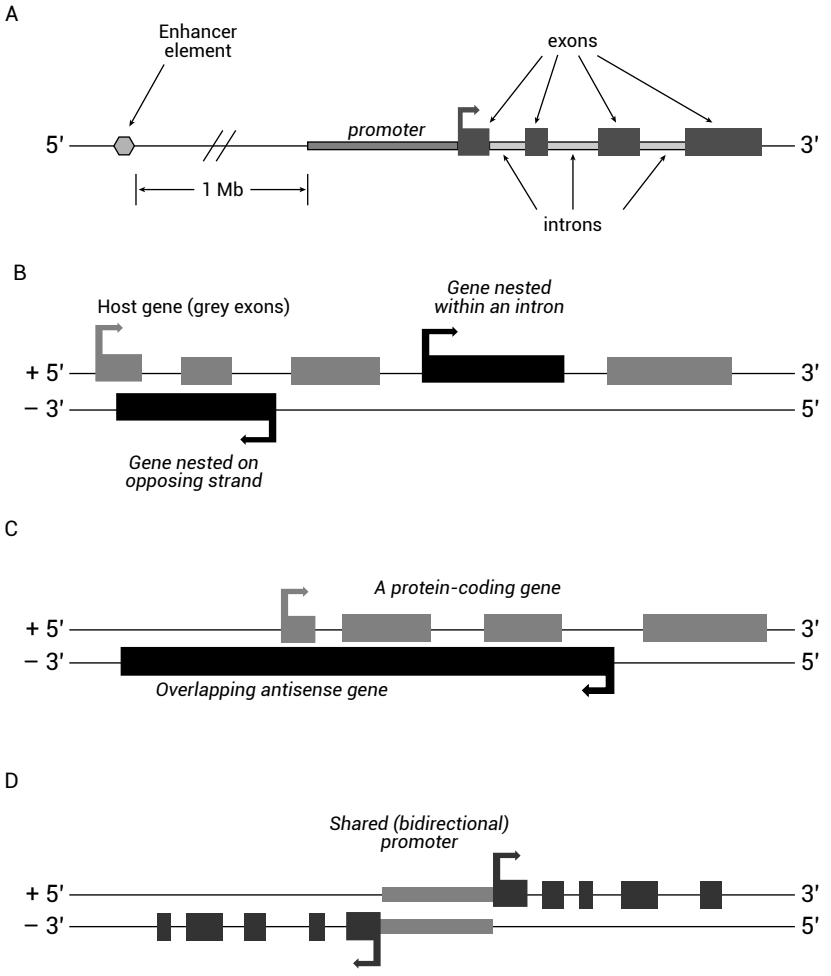

Figure 2. A depiction of why the genes as simple heritable units (marbles in a jar) is not valid in light of our understanding of genomic architecture and the interconnectivity of genes and regulatory elements over large distances. A) The basic structure of a eukaryotic gene representing the 'genes in pieces' concept along with a regulatory connection to a distant enhancer element that would interact with the promoter region of the gene. The arrow in the first exon represents direction of transcription. B) Depiction of two types of nested genes—one running in the same direction as the host gene within an intron and one on the opposing strand. C) Depiction of an overlapping gene—a protein-coding gene and a corresponding antisense long non-coding RNA gene on the opposing strand. D) Configuration of two neighbouring genes on separate strands sharing the same bidirectional promoter.

as evidence for neutrally evolving DNA.[17] As the genomics revolution progressed, it became apparent that the coding regions (exons) of protein-coding genes only occupied less than three percent of the total genome in humans. Because much of the non-coding DNA was not well characterized, it was assumed that it was mostly non-functional and thus subject to Neutral Model evolution. We will refute each of these errant assumptions in turn, based on recent discoveries.

Codon degeneracy refuted

The variability and apparent redundancy in the third base of codons in protein-coding genes was initially termed 'wobble' or 'degeneracy'. The key assumption is that different codon variants in the third base resulting in the same amino acid are functionally equivalent. Thus, it was assumed that mutations which did not change an amino acid in a codon (synonymous) would have no discernable biochemical effect in the cell.

The assumption of codon degeneracy has provided one of the key mechanisms undergirding the Neutral Model for 40+ years. In 2005, Neutral Model advocate Masatoshi Nei stated, "Because of degeneracy of the genetic code, a certain proportion of nucleotide substitutions in protein-coding genes are expected to be silent and result in no amino acid substitution."[10] Nei maintained and reiterated this belief in a follow-up publication reviewing the Neutral Model in 2010 and a book on the subject in 2013.[1,2] However, a number of groundbreaking publications in recent years are completely uprooting this bastion of molecular evolution by providing overwhelming evidence for multi-role biochemical functionality at a codon's third base.[18]

We now know that across the spectrum of life, the genomes of many types of organisms show incredible variability in their preferences for the specific usage of different codons.[19–21] Codon preference for diverse genes has been found to not only differ markedly among diverse prokaryotic and eukaryotic taxa, but also vary widely between different genes even within the same organism's genome.[19,21] The authors of a recent review describing the complicated scenarios of codon usage across the spectrum of life stated that these represent "features that are difficult to explain through mutation alone".[21]

If any seemingly synonymous codon would do, then why the incredible specificity and preference? As it turns out, there are multiple design-based reasons for specificity in codon usage.

In view of the enormous interconnectivity of cellular biochemistry, it makes sense that a specific codon code would be tied to the tRNA production system such that codon differences would control the effectiveness of the protein translation machinery. As the tRNA production levels are 'set' for the original code, codon changes outside this original constraint will cause a tRNA supply imbalance.[22] A more recent discovery has actually shown that tRNAs are reused in the translation process and that codon sequence, particularly at the third base, plays a key role in this cellular recycling system.[23] The tRNA recycling process is especially important for genes that are highly and rapidly expressed to maintain optimal translational efficiency.

Perhaps the greatest refutation of the idea of redundancy has been the discovery of multifunctional codes embedded in the sequences of codons.[24] This idea of multilayered codes within mRNAs derived from genes is not new. It has been demonstrated that protein-coding exons incorporate a variety of signals pertinent to cellular RNA processing machinery, such as splice sites, RNA editing sites, miRNA binding sites, and mRNA turnover signals in addition to the information delineating amino acids.[25] Now it has also been demonstrated in humans that transcription factors commonly clamp onto specific sites encoded within exons inside genes.[26] Incredibly, the same set of codons which specifies a sequence of amino acids also demarcates where transcription factors bind to control and regulate gene transcription.[27] As it turns out, this phenomenon is quite common such that about 14% of codons within 87% of human genes are proven target sites for transcription-factor binding. These dual-function codon sites in the exons of genes are now referred to as 'duons'.

The prevalence of dual multilayered codes in codons creates a severe obstacle for the Neutral Evolution Model—an inconvenient fact that immediately became obvious to scientists after its discovery. Several researchers in a recent paper acknowledged this problem, asking, "How widespread is the phenomenon of 'regulatory' codes that overlap the genetic code, and how do they constrain the evolution of protein sequences?"[28]

Not only does the presence of complex dual codes negate the evolution of proteins via alleged stochastic processes, but it has also recently been demonstrated that the third base of codons plays a key functional role during the production of proteins. As proteins are being translated, occasional pausing occurs while the protein is polymerized and funnelled through a tunnel in the ribosome.[29,30] The sequence delineated in codons dictates the timing of polypeptide pausing as it passes through the ribosome—a process that is critical to the folding and functional three-dimensional shape of the resulting protein. Because the translation and the initial ribosomal-based folding of the protein are integrated together, the operational process is termed 'co-translational'. This translational pausing has now been shown to be controlled specifically by the third base of the codon, adding yet one more overlapping code to the sequence of codons.[31] Once again, the destructive effect of such a discovery on the failing paradigm of evolution was not lost on the researchers as they stated, "The functionality of codonic redundancy denies the

ill-advised label of 'degeneracy'." What was thought for so many years only to be meaningless redundancy and genetic sites for neutrally evolving sequence, has now been proven to be embedded with multilayered codes and critical to cellular function. Evolutionists would say that such sequences would be restrained from evolutionary processes.

Junk debunked

Early studies in reassociation kinetics at the very beginning of the molecular biology era (1970s) seemed to indicate that a large portion of the genome was repetitive in nature, with very little containing the higher complexity of protein-coding regions.[32] When the first draft of the human genome became available in 2001, much of it was found to be difficult to decipher, with less than three percent coding for protein.[33,34] This large undefined fraction was prematurely assigned the label 'Junk DNA', a term that had been used previously, beginning in the early 1970s, to provocatively describe DNA of unknown function as useless evolutionary baggage.[35] These vast non–protein-coding regions of the human genome were immediately thought to be a major source of raw genetic material that could evolve through Neutral Model processes.

As genomics technology began to advance and studies became more comprehensive and sophisticated, researchers began to realize that much more than protein-coding genes were being transcribed into RNA. In fact, nearly the entire genome was eventually found to be transcribed.[36–40] This idea of pervasive transcription inspired some researchers to call the genome an 'RNA Machine'.[41] A significantly large component of this non–protein-coding transcriptional landscape is produced from a diverse class of genes called 'long noncoding RNAs (lncRNA)', which greatly outnumber protein-coding genes by at least two to one.[36–40] The roles that lncRNA transcripts play in the cell are incredibly diverse, ranging from gene regulation, chromatin modification, translational regulation, structural and catalytic components integrated with proteins to intercellular signalling.[24,42–46] Interestingly, many of these lncRNA genes are complexly regulated and spliced similar to protein-coding genes, but are typically expressed at much lower levels and tend to be more specific in their expression to cell state and type.

A limited number of lncRNA genes have been investigated and important function has been assigned to them.[40,42,47–49] While many lncRNA genes have been found to be co-expressed with protein-coding genes or their expression patterns ascribed to specific cell types and states, it has been difficult to assign specific function to many lncRNAs in humans particularly. Of course, large numbers of protein coding-genes in humans still have unknown function. Much of what we know is based on research done on human cells grown in the lab, which are widely studied for the transcription of both protein and non-coding RNA genes and are not necessarily indicative of what goes on inside real bodily tissue.

The Third Way—an extended evolutionary synthesis?

A major reason why an extended evolutionary synthesis, or as some call it 'The Third Way', is gaining ground among secular scientists is that (in one evolutionist's own words) "all the central assumptions of the Modern Synthesis (often called Neo-Darwinism) have been disproved".[50] Of course, evolutionary theory in modern times has never been without its disputes and controversies. Famed vocal evolutionist Douglas Futuyma recently stated this basic truth:

> "Ever since the Evolutionary Synthesis of the 1930s and 1940s, some biologists have expressed doubt that the Synthetic Theory [the prevailing neo-Darwinian version of evolution], based principally on mutation, genetic variation, and natural selection, adequately accounts for macroevolution, or evolution above the species level."[51]

In the heyday of the modern synthesis, prominent evolutionists Ernst Mayr, an authority on speciation and systematics, and George Gaylord Simpson, a leading paleontologist, both inferred from the fossil record that evolution must have occurred erratically in large jumps. This conclusion was based on the realization that transitional fossils were conspicuously lacking and that many fossilized creatures with living counterparts did not appear to have evolved at all. Many fossils, supposedly tens, or even hundreds of millions, of years old are essentially identical to living versions of the same creatures, a fact that evolutionists themselves are troubled over.[52,53]

These glaring evolutionary problems in the fossil record ultimately provided the impetus for the theory of punctuated equilibrium proposed in 1972 by renowned evolutionists Stephen Jay Gould and Niles Eldredge.[54] To accommodate the inconvenient reality of the fossil record and its embarrassing lack of transitional forms, punctuated equilibrium postulates that macroevolution is marked by long periods of stability with no change in morphology (referred to as stasis). This is occasionally interrupted by infrequent bursts of rapid bodily alterations in which a fundamentally new form comes into being. The chief problem with this 'hopeful monster' idea is that the amazing discoveries in molecular biology and genomics that came on the heels of the Punctuated Equilibrium Theory essentially destroyed the molecular genetic foundations of both it and the modern synthesis. The fact of the matter is that all developmental traits are under highly sophisticated, irreducibly complex control involving hierarchical interlocking gene networks, strictly controlled

chromatin states involving non-coding RNAs, histone modifications, DNA methylation, and specific 3-dimensional chromosome conformation and architecture. And all of this nearly infinite complexity dynamically changes according to cell state and type. The number of coordinated beneficial mutations in the genome needed to produce a new 'hopeful monster' is completely improbable.

In the wake of new antievolutionary discoveries in molecular biology and genomics, secular scientists are at odds with each other over how macroevolution can possibly work. Approximately 10 years ago, a dissenting splinter group of prominent evolutionists broke away and formed a movement called The Third Way, or the Extended Evolutionary Synthesis.[50,55–57] In regard to classical neo-Darwinism, the most popular and dominant form of evolution presented in textbooks and the secular mainstream, the Third Way crowd claim that this version of evolution "ignores much contemporary molecular evidence and invokes a set of unsupported assumptions about the accidental nature of hereditary variation".[58] They also go on to state, "The DNA record does not support the assertion that small random mutations are the main source of new and useful variations. We now know that the many different processes of variation involve well regulated cell action on DNA molecules."[58]

So, what does this new daring breed of scientists propose as an alternative model of evolution, given that they also reject the overwhelming evidence that an omnipotent divine engineer is responsible for creating all this 'well regulated cell action'? At present, they are simply taking a position of blissful ignorance and stating that they need "a deeper and more complete exploration of all aspects of the evolutionary process".[58] In other words, no new molecular mechanism for evolution to occur is being proposed, but like Darwin in his day, these scientists are hoping that further scientific discovery will somehow uncover a solution. Of course, this hopeful attitude is despite the fact that progress in molecular biology and genomics is revealing nothing but new layers of irreducible complexity on a regular basis.

The general approach to acquiring more knowledge that would enable some sort of extended evolutionary synthesis has been summed up into four general categories of research in a recent Third Way community report (figure 3).[56] The authors of the paper state, "In this regard, insights derived from research on: (i) evolutionary developmental biology ('evo–devo'), (ii) developmental plasticity, (iii) inclusive inheritance, and (iv) niche construction are particularly instructive." However, as will be discussed below, it is noteworthy that all of these proposed research areas actually present severe problems for the evolutionary model.

Developmental biology along with its organismal specificity of gene networks and extra-chromosomal cellular information, all interacting dynamically together, forms a major hurdle for random evolutionary processes to overcome, as discussed previously in this paper. Creationist researcher Alex Williams also notes that when analyzing essential developmental genes that are often similar in translated protein sequence and gene order among many taxa, no evolutionary explanation of how the toolkit repertoire came to be present at the beginning of animal life can be provided, leaving the conclusion that evolution has played no discernible role at all.[59] Furthermore, the regulatory DNA features and epigenetic mechanisms surrounding the use of developmental gene toolkits is markedly different between different types of organisms and was also present at the beginning of multicellular life.[60,61] While the similarity of sequence in the basic protein-coding regions of some common developmental genes would on the surface seem to support, marked differences between taxa in regulatory sequence structure, differences in overall components of developmental gene regulatory networks, and the organism-specific specificity of overall epigenetic control is a complete evolutionary enigma. These taxonomic differences, combined with the fact that these systems exist at all levels of life, including the alleged beginnings of multicellular life, refute evolution. Intelligent design, however, would predict both

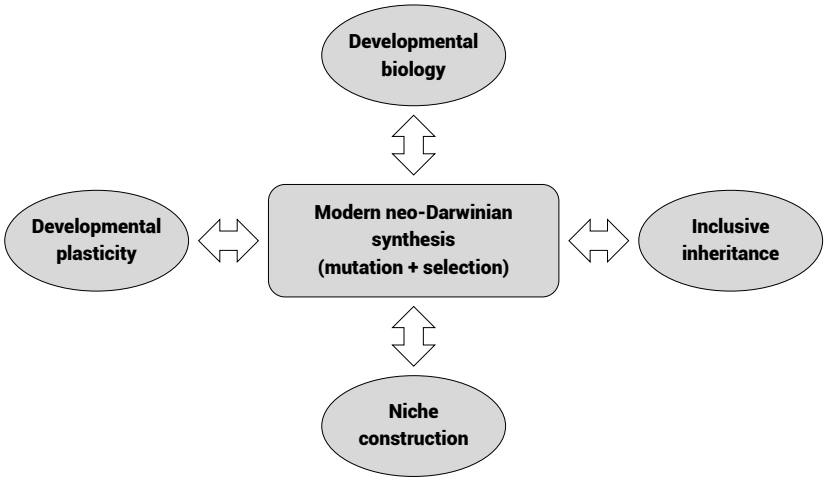

Figure 3. Depiction of the four main areas of research that Third Way proponent evolutionists are hoping will produce results that will enable them to extend the neo-Darwinian synthesis. As described in the text of this paper, these areas of research do not support an evolutionary hypothesis, but rather an intelligent design model of extreme bioengineering by an infinitely powerful and all-wise creator.

pervasive complexity and commonality based on code reuse and engineering principles.

Developmental plasticity is the ability of an organism to modify its development in response to environmental conditions.[62] This involves a complex multilevel system of environmental sensors that constantly monitor and track a diversity of stimuli, resulting in changes in gene expression and cellular physiology. The diversity of stimuli that are tracked and monitored varies widely, depending on the organism and its environment.

One example in animals is that of field crickets, the pregnant mothers of which were exposed to the predation of wolf spiders.[63] These offspring show a heightened response to spider cues, thus surviving better in a spider environment than do crickets, the mothers of which were not exposed. Another more dramatic developmental example is the induction of defensive body structures in water fleas through exposure to predator chemical emissions called 'kariomones'.[64]

The mere presence of these amazingly complex features in living systems not only presents powerful evidence of adaptive systems engineering, but provides great difficulties for the selection model of standard neo-Darwinian evolution, since environmental cues would be acting on something designed to sense them and thus blunt the effects of selection. The major problems that developmental plasticity provides for evolution are not lost on its proponents. By way of example, one of these stated in a recent review paper, "Identifying the factors that promote the origin of complex, novel traits is among the most intriguing and enduring problems in evolutionary biology."[62]

The modern neo-Darwinian synthesis over-simplifies inheritance by reducing it to genes and variations in DNA sequences. In genome-wide association studies it became painfully obvious that most phenotypic traits with high heritability could not be linked to DNA variation in humans.[65] The concept of inclusive inheritance recognizes that biological information is not transmitted across generations by DNA sequence alone, but that both genetic and non-genetic inheritance, and the interactions between them, play interactive roles.[56,65]

Scientists seeking to extend evolution theory recognize that, in addition to genetics, inheritance includes epigenetic, ecological, cultural, and parental factors.[65] Epigenetic inheritance involves a complex array of DNA modifications, histone modifications, and heritable RNAs that are subject to alteration by cellular machinery in response to environmental cues.[66] All of these factors affect development and behaviour, and can even have delayed expression later in life. DNA methylation patterns and histone modifications in particular have been shown to persist over multiple generations. These types of modifications affect development and cellular processes primarily through altering gene expression.

Ecological, cultural, and parental factors are other factors that are heritable. Ecological and cultural factors that persist across generations ultimately effect epigenetic mechanisms and interact with them. In respect to parental factors, genomic imprinting is especially important to mention. Environmental cues, and resulting epigenetic modifications, can cause certain genes to be preferentially expressed as being derived either materially or paternally.[67] Thus, the effect of the environment is complex and responses are based on not only genetics, but the sensor systems and regulatory pathways engineered into organisms. The diversity of factors acting both directly and indirectly on the genome is not only a confounding buffer that negates the mutation-selection paradigm of evolution, but also provides powerful evidence of engineered adaptive mechanisms pointing to a creator.

The last area of research in extending the evolutionary model is that of niche construction—the process in which an organism alters its environment, but not always in a manner that may be conducive to its long-term benefit or survival. An example would be the construction of dams by beavers across rivers and streams. Evolutionists believe that not only does an environment select for changes in an organism, but that organisms cause changes in their environment through niche construction. The obvious complication for the standard evolutionary paradigm is that this back-and-forth scenario creates a complicated feedback relationship between natural selection and niche construction, i.e. that when organisms alter their environment, change can then cause a shift in what traits are being naturally selected for. It's a type of chicken-and-the-egg scenario, but more complicated, since organisms live in communities with other types of organisms that all have some sort of impact on their environment. Really, the environment is not driving any sort of macroevolutionary change but only providing cues that are acted on by sensory and response systems engineered into a wide variety of organisms living in community.

Conclusions

The Neutral Model was an initial effort to attempt to remedy the serious shortcomings of the Neo-Darwinian Theory of evolution. To avoid the problem of directly challenging the reigning paradigm, which would produce enormous opposition to the theory, Kimura once claimed that "neutral theory is not antagonistic to the cherished view that evolution of form and function is guided by Darwinian selection, but it brings out another facet of the evolutionary process by emphasizing the much greater role of mutation pressure and random drift at the molecular level".[4] Although Kimura did not openly deny neo-Darwinism, according to Gould, he views its "processes as quantitatively insignificant to the total picture—a superficial and minor ripple on the

ocean of neutral molecular change, imposed every now and again when selection casts a stone on the waters of evolution".[6]

The Neutral Model incorporates not only codon redundancy, but vast amounts of 'junk DNA' as a source of mutational genetic novelty which forms an inherent assumption of the model. Negating these Neutral Theory assumptions and premises are new discoveries in full codon utility, multilayered embedded codes in and around genes, and pervasive genome transcription and functionality. In addition, extensive computational modelling of Neutral Theory has also revealed that it is defunct as a viable working evolutionary model, and would be even if the genome were heavily composed of 'junk'.

So not only has the Neo-Darwinian Model been disproved, but the alternative Neutral Model has come up wanting as well. The evolutionary response by some has been to reject both evolutionary paradigms along with the obvious conclusion that living systems were engineered by an omnipotent Creator. Their alternative, called the 'Extended Evolutionary Synthesis', is really nothing but a position of blissful ignorance in hopes of discovering some yet unknown evolutionary process in a variety of research areas that are, in reality, only proving to be goldmines of opportunity for creation scientists. Scientific discovery in the area of molecular biology and genomics is steam rolling forward and only revealing a picture of nearly infinite cellular and organismal complexity.

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Jeffrey P. Tomkins *has a Ph.D. in Genetics from Clemson University, an M.S. in Plant Science from the University of Idaho, Moscow, and a B.S. in Agriculture Ed. from Washington State University. He was on the Faculty in the Dept of Genetics and Biochemistry, Clemson University, for a decade, where he published 58 secular research papers in peer-reviewed scientific journals and seven book chapters in scientific books—in the area of genetics, genomics, and proteomics. For the past five years, Dr Tomkins has been a Research Scientist at ICR where he has published 20 peer-reviewed creation science journal papers, numerous semi-technical articles on the ICR web site and their magazine* Acts & Facts*, and two books.*

Jerry Bergman *has nine academic degrees, including 5 masters and two PhDs. His major areas of study for his graduate work include anatomy and physiology, biology, chemistry, and psychology. He has graduated from Wayne State University in Detroit, Medical University of Ohio in Toledo, University of Toledo and Bowling Green State University. A prolific writer with over a thousand publications to his credit, including 43 books and monographs, Dr Bergman has taught biology, microbiology, anatomy and physiology, chemistry, biochemistry, geology, astronomy and psychology at the college level. Now retired, he has taught at The University of Toledo Medical College, The University of Toledo, Bowling Green State University and other schools for a total of close to 50 years.*