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New Genetic Information Proposals Fail

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A major problem in evolutionary dogma is the origin of new genetic information. Getting new genetic information is required to go from a single-celled organism to a person. A bacterium, for example, simply does not have the same information or as much as a human does in its genome. In order to get to a person from a bacterium, among many other things, new information must be introduced—and a lot of it.

Evolutionary scientists know they need to explain the origin of genetic information. However, instead of discussing new information, they tend to focus on new genes. These are sometimes known as de novo genes. In the literature, they have proposed different methods to create these "new genes" or new expressions of genes, but only four are well accepted, and we will discuss those below. Extensive research is underway in these areas, and hundreds of papers are published yearly on these topics. However, their methods are rarely empirical and are drawn largely from theory rather than evidence.

Gene Duplication

Since gene duplications are rarely observed, they are often postulated post hoc by looking at phylogenetic trees.

Likely the most popular explanation the evolutionists use to describe the existence of de novo genes is gene duplication. Gene duplication is not limited to a single gene. According to the theory, sometimes even whole genomes can be duplicated. Most duplications are not that large, consisting of a single gene or piece of a gene. Generally, the duplication occurs due to either a replicated mobile element in the genome (a gene that has the ability to move) or an error during recombination (rearrangement of genes that takes place during meiosis (Panchy et al. 2016). Since gene duplications are rarely observed, they are often postulated post hoc by looking at phylogenetic trees (Wapinski et al. 2007). One of the traits supposed to have arisen from gene duplication is C4 photosynthesis in plants (Flagel and Wendel 2009).

While duplications do occur, they do nothing to help evolutionists in their pursuit of new genetic information. A duplication is analogous to getting a second instruction manual for a car. The extra copy is just extra junk in the glove box that will likely never be opened, let alone read. Even were it to be read, nothing in the second copy would differ from the first copy.

Evolutionists are aware the second copy has no purpose when it arises, so they propose that it can be repurposed. Repurposing the newly duplicated genes can occur in a number of different ways. One possible fate of a duplicated gene (or group of genes) is something called "gene conservation." According to theory, when this happens the genome keeps both the old copy and the duplicated copy. According to evolutionary phylogenetic studies, gene conservation can be common (Feuda et al. 2016). However, theory disagrees with phylogenetic studies because the only way to maintain both genes as functional would be for both to maintain the same mutation rate, which evolutionists view as unlikely (Krakauer and Nowak 1999). Thus gene conservation is likely rare and not a robust explanation. This is especially true given the tendency of newly formed polyploids to undergo rapid gene loss after formation (Clarkson et al. 2005). Thus gene conservation appears to contradict other aspects of the evolutionary model.

The outcome the evolutionists really want to talk about—and need to have happen for gene duplication to work—is neofunctionalization. *Neofunctionalization* is the process that purportedly creates new functional genetic information in the duplication. This process would cause the organism to get something truly new, like a new structure or new function, to an existing protein. Neofunctionalization is rarely, if ever, observed. There are reams of papers talking about neofunctionalized genes, but almost all of them rely heavily on phylogenetics to make their arguments (Lynch 2007). Neofunctionalization is so rare, even some evolutionists have questioned its existence, pointing instead at subfunctionalization (Gibson and Goldberg 2009).

Subfunctionalization only works if the original gene had more than one function. If the original gene was multi-functional, subfunctionalization causes the functions to be split between the original and duplicate genes. Evidence for this, like neofunctionalization, is spotty at best. As an example, one paper assumed the evolutionary story was true and thus poplars and mangroves are related despite not being in the same family. The paper used this likely false claim to argue that one gene in their common ancestor had merged and been subfunctionalized in mangroves but had split again in poplars (Cusak and Wolfe 2007). The paper more closely resembled a flight of fancy with a genetics veneer than it did science.

The paper used this likely false claim to argue that one gene in their common ancestor had merged and been subfunctionalized in mangroves but had split again in poplars! The paper more closely resembled a flight of fancy with a genetics veneer than it did science.

Even without the absurd phylogenetic storytelling, at least some evolutionists have realized that natural selection has no ability to "create" subfunctionalized genes. Genes are under tight regulatory control and rarely change expression, even after the whole genome is duplicated as is the case in polyploidy (Rogers, Shao, and Thornton 2017). This fact has caused some evolutionists to claim that subfunctionalization must eventually proceed to neofunctionalization (Rastogi and Liberles 2005). However, the only way to do so is by mutations, most of which are negative (Mezmouk and Ross-Ibarra 2014). Thus, even if the evolutionists are right and subfunctionalization does occur, a subfunctionalized gene would be more likely to be destroyed by a deleterious mutation than neofunctionalized by a beneficial one.

The last potential fate of a duplicated gene is the one that is most common and least popular among evolutionists: nonfunctionalization. Nonfunctionalization occurs when a mutation breaks one of the copies of the duplicated gene so that it no longer functions as intended. According to evolutionary ideas, because there are two copies of the gene, the mutation is not deleterious and the broken gene becomes a pseudogene (Zhang 2003). A pseudogene is a broken gene that sometimes produces noncoding RNA and sometimes is merely genetic baggage or "junk DNA." Evolutionists claim pseudogenes largely have no function. However, evidence is beginning to mount that pseudogenes have a function in the genome (Cheetham, Falkner, and Dinger 2020).

If pseudogenes have function, the theory behind many nonfunctionalization events gets messy. Pseudogenes are supposed to be created by neutral mutations. However, most mutations are deleterious (Cowperthwaite, Bull, and Meyers 2006), making it quite problematic to generate new functional pseudogenes using mutations. What that means in practice is that nonfunctionalization likely breaks genes that can potentially be removed. It does not create functional pseudogenes.

Because the gene duplication story is loaded with contradictions and weaknesses, it is difficult to envision it producing new genetic information. Gene duplication has a number of crippling problems from an empirical science perspective which, taken together, rule it out as a source of genetic information (Bergman 2006). Without new information, there can be no new structures or functions.

Internal Gene Duplication

Internal gene duplication is similar, in a way, to gene duplication, with one major difference. Only part of a single gene is duplicated and thereafter inserted into the gene or attached to the end. Such an event extends the gene. Like gene duplications, most of the internal gene duplications are hypothesized from a phylogenetic tree (Lawson, Charlebois, and Dillon 1996) or by looking at similar repeated sequences in different organisms' genomes (Barker, Ketcham, and Dayhoff 1978; Chen, Li, and Sung 2007).

There is, however, empirical evidence of internal gene duplications. It just is not the kind of evidence evolutionists expected or want. For example, internal gene duplication in the FLT3 gene is linked with a high risk for leukemia (Abu-Duhier et al. 2000). Internal gene duplications are also tied to BRCA1 gene-related cancers (Willis et al. 2017). It seems that internal gene duplications tend to break genes and cause disease.

Even if an internal gene duplication was not deleterious, they do not produce new information.

Even if an internal gene duplication was not deleterious, they do not produce new information. As an analogy, consider the sentence: "Mutations produce no new genetic information: they merely break something that is already there." If we were to take a chunk of that sentence, duplicate it, and drop it on the end of the sentence, would there be any new information present? "Mutations produce no new genetic information: they merely break something that is already there genetic information: they merely." Has the sentence been improved? Absolutely not! It is the same with DNA. The sequences already code for RNA. Adding something in the middle or end is likely to turn the instructions into nonsense. In other words, the gene would become nonfunctionalized rather than generating new function.

Exon Shuffling

Exon shuffling is more technical than gene duplication and requires the use of technical terms. However, in simplified terms, before the RNA strand is translated, part of what was transcribed is removed and not translated. The excess pieces are referred to as introns. The leftover part is composed of exons. Each exon codes for a section, or a domain, or a protein.

According to the evolutionists, exon shuffling occurs two ways. Either an exon is duplicated and the copy moved to a new place in the gene, or sometimes an exon gets moved to an entirely new gene. There is, however, a problem. Moving an exon around has a very high likelihood of breaking something termed the reading frame. In other words, it would change the way the gene is read, likely breaking it. Only a small subset of exons, termed symmetric exons, do not change the reading frame (Kolkman and Stemmer 2001).22 Given that a broken reading frame likely breaks the gene, it is not surprising that the majority of the exon-shuffling events evolutionists propose are symmetric (França, Cancherini, and de Souza 2012).

Evolutionary dogma argues that exon shuffling moves exons that correspond to protein domains into new genes, thereby creating new proteins or adding domains to existing proteins (Silverman et al. 2006). The evidence presented for this position is largely phylogenetic (Long et al. 1996). Generally this is done through the appeal to sequence homologies (Babushok et al. 2007).

Exon shuffling does occur, but it does not seem to do what the evolutionists want it to do. Generally, exon shuffling occurs as a result of illegitimate recombination. Normal recombination is carefully controlled by the cell and helps maintain genotypic and phenotypic diversity. Illegitimate recombination involves crossing over between genes that are not the same. Sometimes, the genes do not even share the same chromosome. Illegitimate recombination is associated with diseases like Duchenne's muscular dystrophy (van Rijk and Bloemendal 2003). Disease association is a common theme of the mechanisms evolutionists propose for creating genetic information.

Alternative Splicing

Alternative splicing is final process that evolutionists propose to create new information in the genome. This is the method with the strongest empirical support, but it still does not do what evolutionists want it to do. Alternative splicing allows the genome to read a gene multiple ways. In other words, the cell transcribes the DNA of a gene, then splices out different exons depending on the protein it is making. Sometimes this happens even before the creation of the mRNA strand (Kelemen et al. 2013). A sizeable majority of the human genome is alternatively spliced (Modrek and Lee 2002), and the average gene has up to three different splicing options (Stamm et al. 2005). However, alternative splicing does not create new genetic information

Different alternative splicing does not produce new information. When splicing errors occur, they cause disease.

Because it allows the same gene to be read multiple ways, alternative splicing allows the genome to be kept much smaller while producing the same number of proteins. That looks much more like a design feature than a product of chance. Even if we grant evolution the benefit of the doubt and assume alternative splicing arose by chance, there is still a problem. Different alternative splicing does not produce new information. When splicing errors occur, they cause disease (Tazi, Bakkour, and Stamm 2009). No new beneficial information is added. The information is already there. Alternative splicing simply allows the genome to combine the information differently.

New Information Lacking

While evolutionists have proposed a number of mechanisms to generate new information, none of them do what is claimed. Instead, they either break the genome or rearrange existing information. Even if the mechanisms did not cause disease, simply creating new sequences are not enough. The new sequences must be able to be read and not create mutations, which are almost exclusively deleterious. Even if the required genetic sequences could be generated, a significant number of beneficial mutations would be required to create new functional information. Evolution simply lacks the mechanism required to create new information.

References

Abu-Duhier, F.M., A.C. Goodeve, G.A. Wilson, M.A. Gari, I.R. Peake, D.C. Rees, E.A. Vandenberghe, P.R. Winship, and J.T. Reilly. 2000. "FLT3 Internal Tandem Duplication Mutations in Adult Acute Myeloid Leukaemia Define a High-Risk Group." *British Journal of Haematology* 111, no.1 (October): 190–195.

Babushok, Daria V., Kazuhiko Ohshima, Eric M Ostertag, Xinsheng Chen, Yanfeng Wang, Prabhat K Mandal, Norihiro Okada, Charles S. Abrams, and Haig H. Kazazian Jr. 2007. "A Novel Testis Ubiquitin-Binding Protein Gene Arose by Exon Shuffling in Hominoids." *Genome Research* 17, no.8 (August): 1129–1138.

Barker, Winona C., L.K. Ketcham, and M.O. Dayhoff. 1978. "A Comprehensive Examination of Protein Sequences for Evidence of Internal Gene Duplication." *Journal of Molecular Evolution* 10, no.4 (February 21): 265–281.

Bergman, Jerry. 2006. "Does Gene Duplication Provide the Engine for Evolution?" *Journal of Creation* 20, no.1 (April): 99–104.

Pancy, Nicholas, Melissa Lehti-Shiu, and Shin-Han Shiu. 2016. "Evolution of Gene Duplication in Plants." *Plant Physiology* 171, no. 4 (August): 2294–2316.

Cheetham, Seth W. Geoffrey J. Faulkner, and Marcel E. Dinger. 2020. "Overcoming Challenges and Dogmas to Understand the Functions of Pseudogenes." *Nature Reviews Genetics* 21 (17 December): 191–201.

Chen, Chun-Chang, Wen-Hsiung Li, and Huang-Mo Sung. 2007. "Patterns of Internal Gene Duplication in the Course of Metazoan Evolution." *Gene* 396, no. 1 (July 1): 59–65.

Clarkson, James J., K. Yoong Lim, Ales Kovarik, Mark W. Chase, Sandra Knapp, and Andrew R. Letich. 2005. "Long-Term Genome Diploidization in Allopolyploid *Nicotiana* Section *Repandae* (Solanaceae)." *New Phytologist* 168, no. 1 (October): 241–252.

Cowperthwaite, Matthew C., J.J.Bull, and Lauren Ancel Meyers. 2006. "From Bad to Good: Fitness Reversals and the Ascent of Deleterious Mutations." *PLOS Computational Biology* 2, no. 10 (October 20): e141.

Cusack, Brian P., and Kenneth H. Wolfe. 2007. "When Gene Marriages Don't Work Out: Divorce by Subfunctionalization." *Trends in Genetics* 23, no. 6 (June): 270–272.

Feuda, Roberto, Ferdinand Marlétaz, Michael A. Bentley, and Peter W.H. Holland. 2016. "Conservation, Duplication and Divergence of Five Opsin Genes in Insect Evolution." *Genome Biology and Evolution* 8, no. 3 (February 9): 579–587.

Flagel, Lex E., and Jonathan F. Wendel. 2009. "Gene Duplication and Evolutionary Novelty in Plants." *New Phytologist* 183, no. 3 (August): 557–564.

França, Gustavo S., Douglas V. Cancherini, and Sandro J. de Souza. 2012. "Evolutionary History of Exon Shuffling." *Genetica* 140, nos. 4–6 (June): 249–257.

Gibson, Todd A., and Debra S. Goldberg. 2009. "Questioning the Ubiquity of Neofunctionalization." *PLOS Computational Biology* 5, no. 1 (January): e1000252.

Kelemen, Olga, Paolo Convertini, Zhaiyi Zhang, Yuan Wen, Manli Shen, Marina Falaleeva, and Stefan Stamm. 2013. "Function of Alternative Splicing." *Gene* 514, no. 1 (February 1): 1–30.

Kolkman, Joost A., and Willem P.C. Stemmer. 2001. "Directed Evolution of Proteins by Exon Shuffling." *Nature Biotechnology* 19, no. 5 (May): 423–428.

Krakauer, David C., and Martin A. Nowak. 1999. "Evolutionary Preservation of Redundant Duplicated Genes." *Seminars in Cell and Developmental Biology* 10, no. 5 (October): 555–559.

Lawson, F.S., R.L. Charlebois, and Jo-Anne R. Dillon. 1996. "Phylogenetic Analysis of Carbamoylphosphate Synthetase Genes: Complex Evolutionary History Includes an Internal Duplication within a Gene which can Root the Tree of Life." *Molecular Biology and Evolution* 13, no. 7 (September): 970–977.

Long, Manyuan, Sandro J. De souza, Carl Rosenberg, and Walter Gilbert. 1996. "Exon Shuffling and the Origin of the Mitochondrial Targeting Function in Plant Cytochrome c1 Precursor." *Proceedings of the National Academy of Sciences USA* 93, no. 15 (July): 7727–7731.

Lynch, Vincent J. 2007. "Inventing an Arsenal: Adaptive Evolution and Neofunctionalization of Snake Venom Phospholipase A2 Genes." *BMC Evolutionary Biology* 7, no. 1 (January 18): 2.

Mezmouk, Sofiane, and Jeffrey Ross-Ibarra. 2014. "The Pattern and Distribution of Deleterious Mutations in Maize." *G3: Genes, Genomes, Genetics* 4, no. 1 (January 10): 163–171.

Modrek, Barmack, and Christopher Lee. 2002. "A Genomic View of Alternative Splicing." *Nature Genetics* 30, no. 1 (January): 13–19.

Rastogi, Shruti, and David A. Liberles. 2005. "Subfunctionalization of Duplicated Genes as a Transition State to Neofunctionalization." *BMC Evolutionary Biology* 5, no. 28 (April 14). doi:10.1186/1471-2148-5-28.

Rogers, Rebekak L., Ling Shao, and Kevin R. Thornton. 2017. "Tandem Duplications Lead to Novel Expression Patterns Through Exon Shuffling in *Drosophila yakuba*." *PLOS Genetics* 13, no. 5 (May 22): e1006795.

Silverman, Joshua, Qiang Liu, Alice Bakker, Wayne To, Amy Duguay, Ben M Alba, Richard Smith, et al. 2005. "Multivalent Avimer Proteins Evolved by Exon Shuffling of a Family of Human Receptor Domains." *Nature Biotechnology* 23, no. 12 (December): 1556–1561.

Stamm, Stefan, Shani Ben-Ari, Ilona Rafalska, Yesheng Tang, Zhaiyi Zhang, Debra Toiber, T.A. Thanaraj, and Hermona Soreq. 2005. "Function of Alternative Splicing." *Gene* 344 (3 January): 1–20.

Tazi, Jamal, Nadia Bakkour, and Stefan Stamm. 2009. "Alternative Splicing and Disease." *Biochimica et Biophysica Acta* 1792, no. 1 (January): 14–26.

van Rijk, Anke, and Hans Bloemendal. 2003. "Molecular Mechanisms of Exon Shuffling: Illegitimate Recombination." *Genetica* 118, nos. 2–3 (July): 245–249.

Wapinski, Ilan, Avi Pfeffer, Nir Friedman, and Aviv Regev. 2007. "Natural History and Evolutionary Principles of Gene Duplication in Fungi." *Nature* 449, no. 7158 (6 September): 57–64.

Willis, Nicholas A., Richard L. Frock, Francesca Menghi, Erin E. Duffey, Arvind Panday, Virginia Camacho, E. Paul Hasty, Edison T. Liu, Frederick W. Alt, and Ralph Scully. 2017. "Mechanism of Tandem Duplication Formation in *BRCA1* Mutant Cell." *Nature* 551, no. 7682 (22 November): 590–595.

Zhang, Jianzhi. 2003. "Evolution by Gene Duplication: An Update." *Trends in Ecology and Evolution* 18, no. 6 (June): 292–298.