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TASC

TASC's mission is to rebuild and strengthen the foundation of the Christian faith by increasing awareness of the scientific evidence supporting the literal Biblical account of creation and refuting evolution.

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Does the Fact That Random Genetic Mutations Can Result in Adaptation Prove Macroevolution? (Part 2)

By Dan W Reynolds, PhD

This article is the second part of a review of Michael Behe's new Book *Darwin Devolves*. The first part covered chapters 1–6.[1] This article will complete the review and covers chapters 7–10.

Chapter 7: Poison-Pill Mutations

Behe observes that biological radiations[2] are only at the species/genus level. He offers three reasons for this. First, species have many features that are irreducibly complex on several levels. If any of these features were to be modified significantly, they would cease to function. Since these features need all their parts correctly fashioned and in place to facilitate functionality, a stepwise mechanism that gradually shaped and cobbled the parts together is extremely improbable. Likewise, random modifications to an irreducibly complex system will result in loss of function long before a new coherent functional system can be assembled and selected. Second, random mutations are more likely to break things than haphazardly construct new beneficial systems. Last, natural selection is likely to favor maintaining an organism in its present form. Since most adaptive mutations are also deleterious to genetic information, an evolving population will eventually run out of things to break, leaving it brittle and relatively inflexible to additional environment stress. Hence Darwinian evolution is self-limiting. The Darwinian mechanism works for microevolution but is incapable of the innovation required for macroevolution.

Behe then discusses the power of random mutations. He cites a twenty-five yearlong experiment by Michigan State University professor Richard Lenski with *E. coli* bacteria. At the time Behe wrote the book (2019), the experiment had produced about 65,000 generations of bacteria. Lenski grew cultures in twelve flasks. The bacteria would go through six to seven generations per day per flask. Each culture contained hundreds of millions of cells. Each day, a one percent sample was transferred to a new flask. The process was repeated daily. Because of the population sizes and rates of reproduction, Behe says that the experiment was equivalent to a million years of evolution of a large animal. Hence the experiment had everything going for it to examine the creative powers of evolution: large populations, rapid reproduction, repeated isolation of small populations, etc. The twelve cell lines acquired different mutations, as would be expected if the mutations are truly random. Lenski noted that some of the cell lines began to reproduce more rapidly after a few hundred generations. Lenski was collecting samples every few hundred generations and freezing them for later comparative studies. The acceleration in the rate of reproduction increased rapidly at first but then slowly declined. The faster reproducing cells had also become larger in volume. Increases in cell culture growth rate came in discrete steps. Once a rapid replicator was produced, it quickly took over the population. Some cell lines grew faster with alternative food sources, some did not. Eventually, five of the twelve cell lines lost the ability to repair their DNA, increasing the mutation rates a hundred-fold. All the cell lines eventually replicated faster and lost their ability to metabolize ribose. DNA studies revealed that mobile viral elements near the genes that enabled ribose metabolism had been deleted in all twelve cell lines. In addition, they found that chunks of the DNA for the genes controlling ribose metabolism had also been deleted. The loss of ribose metabolism was shown to increase the cell replication rate by one to two percent. Hence a degradative mutation had resulted in better survival. Apparently, the resources used to facilitate ribose metabolism were better used elsewhere under the circumstances. Other broken genes were found,

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[1] Reynolds DW. Does the fact that random genetic mutations can result in adaptation prove macroevolution? *TASC Newsletter*, January 2020. <https://tasc-creationscience.org/article/does-fact-random-genetic-mutations-can-result-adaptation-prove-macroevolution>. Accessed 2020 Jan 28.

[2] Radiations are alleged periods of rapid evolutionary change as inferred from the fossil record.

some of which controlled formation of the cell membrane, perhaps explaining why the rapidly replicating cells increased in volume.

A total of fifty-nine genes had changed their activity in the "evolved" cells. Additional mutations were found in a regulatory gene, called spoT, which controls emergency responses at the onset of starvation. The mutations in spoT resulted in changing the activity levels of fifty-nine downstream genes. Although the mutations in spoT differed in the various cell lines, the effect was the same in eight of the twelve cell lines. The fact that the same regulatory response resulted from various mutations suggests that the various point mutations had damaged the regulatory protein's functionality in a similar way; there are many ways to break things. Hence the increased activity of fifty-nine genes in eight cell lines that resulted in improved adaptation arose by damaging a regulatory gene with various point mutations.

In 2016, Lenski reported 264 complete genomes of *E. coli* from several generations spanning the entire experiment. The paper had two tables of the genes which were most often mutated. In the first table, fifteen genes were listed which had mutations in multiple places, suggesting the genes had been damaged. In the second table, there were 16 genes that had suffered repeated insertions or deletions, modifications that usually kill genes. Behe summarizes:

> The bottom line is this. After fifty-thousand generations of the most detailed, definitive evolution experiment ever conducted, after so much improvement of the growth rate that descendant cells leave revived ancestors in the dust, after relentless mutation and selection, it's very likely that all of the identified beneficial mutations worked by degrading or outright breaking of the respective ancestor genes. And the havoc wreaked by random mutation had been frozen in place by natural selection.[3]

Behe wrote a review article in 2010 that explored the nature of beneficial mutations observed primarily in microbes.[4] The vast majority of the beneficial mutations cited involved loss of function in some molecular system. These results underscored Behe's First Rule of Adaptive Evolution, that most beneficial mutations result from destroying biological information. Insertions and deletions are "frame shift" mutations that almost always result in an incorrect protein. Point mutations often degrade or destroy a protein's function. There may be only a handful of beneficial mutations that are also accompanied by a gain in function. Even then, the gain in function may still have a downside (e.g. sickle cell anemia). In contrast, there are hundreds to thousands more beneficial mutations that result from damaging some molecular entity. And this is why Darwinian macroevolution does not work: beneficial yet damaging mutations will be fixed long before the few beneficial and constructive mutations are ever found. Organisms which adapt by destroying information become less and less flexible to changing environments.

Some of the cell lines in Lenski's experiments started metabolizing citrate. The genes for the proteins required for citrate metabolism were already there. However, those genes are not typically activated except under anerobic conditions. A regulatory gene had mutated leaving the switch that controlled citrate metabolism permanently "on." So, these bacteria could now metabolize citrate in the presence of oxygen, but at the cost of the function of a regulatory gene. These "citrate eaters" had also lost the ability to metabolize ribose, had a diminished ability to proof-read DNA during replication, and had lost the function of a dozen other genes as well.

A 2013 study looked at adaptations of bacteria to 144 different environmental conditions. In ninety-six percent of the conditions, the bacteria increased their growth rate by breaking a gene.

Behe briefly discussed other examples of adaptation through breaking genes related to clover, horses, and people.

Chapter 8: Dollo's Timeless Law

Evolution through adaptation by degradative mutations inevitably results in organisms which are less able to adapt to changing environments. They become brittle and hence self-limiting; they can change within the species/genus boundary, but no further.

Historically, natural selection became a designer substitute. But natural selection only selects what is advantageous now; it has no ability to plan, no foresight, no way to select successive mutations with a view to building something in a coherent way. Lenski's experiments showed what evolution does over thousands of generations: adaptation through degradation.

It is currently believed by evolutionists that new proteins evolve by gene duplication followed by neutral mutations in the duplicate until a new functional protein emerges.

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[3] Behe MJ (2019) *Darwin Devolves*, Harper One, New York, NY, 179

[4] Behe MJ (2010) Experimental evolution, loss-of-function mutations, and "the first rule of adaptive evolution." *Q Rev Biol* 85(4): 419-445. This article is available for free at https://www.researchgate.net/publication/49764025.

Hence proteins with similar functions and amino acid sequences allegedly share a common ancestor gene. Two steroid receptor proteins, MR and GR, are thought to have emerged through the gene duplication mechanism. MR binds with steroids A, B, and C while GR only binds to steroid C. Each protein folds in a specific way as dictated by the location of electrostatic forces in their amino acid sequences. The folded proteins take on specific shapes which enable them to bind to specific steroids. In the cell, binding a steroid by MR and GR results in signaling the cell to perform specific tasks. Researchers wanted to see if they could synthesize the ancestral gene and then find the mutational pathway to the extant MR and GR proteins. By comparison of the genes for MR and GR, the structure of the alleged ancestral gene was inferred. The scientists made the inferred gene and then put into bacteria. It produced a protein with much of the binding characteristics of MR, but not GR. Hence, they set out to find a mutational pathway to a gene which would produce a protein with binding characteristics similar to GR. The researchers found a specific sequence of two specific mutations that produced a protein which behaved like GR. The order of the mutations was critical as only one of the two mutational pathways permitted retention of function at each step. Hence, they showed how an alleged ancestral protein *might* have evolved into two proteins that bind steroids differently. They did not show that this pathway would actually be traversed. In addition, they did not explain the origin of the amino acid sequences needed to bind the steroids in the first place. Also, no new binding interactions evolved; the abilities to bind to steroids A, B, and C were present from the beginning. For GR to form, the alleged ancestral protein would have had to lose its ability to bind to steroids A and B. Also, their experiments shed no light on how the molecular systems controlled by MR and GR emerged or the origin of the steroids. At best, the work showed that there is a potential pathway to generate two different proteins by the gene duplication/mutation/selection mechanism in this instance. But again, no new steroid/protein interactions evolved nor was the origin of the three protein/steroid interactions explained. And, of course, the whole scenario was speculative since they had no way of knowing what the ancestral genes for MR and GR were or even if those genes ever existed. Behe considered the results of their study modest insofar as their demonstrating the power of the Darwinian mechanism to evolve novel information. Behe had shown previously that changes requiring two specific mutations were within the range of Darwinian processes if the population size, mutation rate, and generation times were favorable.[5]

The researchers that had examined the possible evolution of MR and GR from a common ancestor protein then wanted to see if GR could evolve back into the alleged ancestral form by the mutation/selection process. The researchers' conclusion, after much experimental work, was that the "reverse evolution" was not feasible by a Darwinian process. Extant GRs are so different from the alleged ancestor protein that it would require too many specific mutations to enable binding with several steroids. In other words, GR would most likely mutate into a useless protein long before the mutations required to impart the desired steroid binding affinities could accumulate. Behe holds this up as an example of the irreversibility of adaptation by Darwinian processes (Dollo's Law). Hence proteins are likely stuck in their present roles. If a protein is well suited to its present task, natural selection will most likely favor it retaining its function and structure rather than breaking it for the sake of some distant future function. So, Dollo's Timeless Law states that natural selection can't plan or coordinate successive mutations towards the construction of a complex machine; it does not know what came before or how to plan for the future—it only selects what is beneficial now.

Behe stresses the importance of experimentation. What may seem straightforward in principle may be very difficult in practice. Every alleged evolutionary step must be backed with experiments. Behe recalls his Principle of Comparative Difficulty: if simple things are hard, complex things are very difficult if not impossible. Behe believes that proteins that require one or two mutations to evolve may be accessible by the Darwinian mechanism, but anything requiring more mutations is very unlikely. He says that molecular machines made out of several proteins, each of which may need to adopt a specific shape, and hence amino acid sequence, in order to interact with the other proteins, is extremely unlikely.

Behe wonders why so many biologists are still so confident in Darwinian evolution. There are still no detailed explanations for the origin of complex molecular machines. Behe says many biologists naively believe Darwinian processes can deliver what has been attributed to them. Confident pronouncements that Darwinian processes can account for the marvels of life is just so much bluster.

Damaged genes may make an organism better suited to a particular environment, but this does not explain where the genes came from in the first place. Population genetics can explain gene frequencies but not the origin of the genes.

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[5] Behe MJ (2007) *The Edge of Evolution: The Search for the Limits of Darwinism*, Free Press, New York, NY.

Chapter 9: Revenge of the Principle of Comparative Difficulty

Random mutations and natural selection are self-limiting. The evolution of the human eye seemed trivial to Darwin because he was unaware of the molecular basis of life. We now know that all explanations for evolution must be on the molecular level. Any evolutionary explanations must include step-by-step descriptions of the syntheses of molecules, the construction of molecular machines, the coordination of the construction and function of numerous molecular machines, the origin of the regulatory apparatus for the construction and function of the various interacting molecules and molecular machines, etc. For macroevolution, it must be experimentally demonstrated that complex functional integrated molecular machinery can continue to function while being modified in ways that promote new functions and organization.

Behe says that there is still no evidence that irreducibly complex molecular systems could ever be built by the random mutation/natural selection mechanism. Moreover, Behe says that all complex molecular machines made of several protein parts are *comprehensively complex*. By this he means that irreducible complexity is not only found at the level of the machine but also in its parts. The proteins that make up a molecular machine must have the correct amino acids in the right places in order for the protein to adopt the correct shape for its role in the machine. Behe cites the molecular machine called gyrase as an example.

Behe then introduces *mini-irreducible complexity* (mIC). He gives as an example the formation of a disulfide bond, often found in proteins. Disulfide bonds consist of a sulfur-sulfur bond formed between two cysteine amino acids. In order for the disulfide bond to form, the two cysteines must be in close enough proximity. Their proximities depend on the amino acid sequence and the overall shape of the protein once it has folded. Usually, many mutations to an existing protein are required before a new selectable function appears. A new feature that required a disulfide linkage would require mutations that generated two cysteine residues in the right locations. All this would have to happen by chance in a stepwise fashion before natural selection could fix the changes.

Behe has calculated how many generations would be required to generate two, three, and four specific mutations required for the emergence of a new selectable function. Behe assumed the gene duplication/mutation/selection mechanism. Behe considered several factors, including the DNA mutation rate and population size. Behe concluded that a billion generations would be required to produce a selectable feature requiring two mutations. A critic of Behe's, starting with assumptions more friendly to evolution, calculated 100 million generations would be needed. Features requiring three or more mutations would require exponentially more generations. Selectable features requiring one mutation without gene duplication would require ten thousand generations. The problem, says Behe, is that damaging yet adaptive point mutations will always vastly outnumber all other types of mutations, so they will always dominate; there are always many more ways to break things than to build them. Hence a functional gene or its duplicate will almost always become nonfunctional before a new selectable feature can emerge. And if the Darwinian mechanism has trouble accounting for mIC systems requiring two mutations, how can it account for molecular machines, various cell and tissue types, various organs, long and complex biochemical pathways, etc.?

According to Behe, macroevolution has three hurdles to overcome: random mutations, natural selection, and irreducible complexity. Random mutations tend to damage things. Natural selection keeps adaptive yet damaging mutations. New irreducibly complex systems need multiple specific coordinated mutations, which take a long time, during which time the random mutation/natural selection mechanism will be hard at work doing its damage. Behe estimates it takes a million times longer to produce one mIC system from two mutations than a one adaptive yet damaging mutation.

Chapter 10: A Terrible Thing to Waste

Random mutations and natural selection lead to adaptation at the cost of damaged genetic information. The process eventually leaves an organism with little left to break and hence less adaptability to future environmental stress. The random mutation mechanism has no foresight, so it will work to find the fastest and shortest solution. In so doing, better more adaptive and even constructive changes that require coordinated mutations are never, or very rarely ever, realized.

Materialists insist that the mind is an illusion—only an automatic nervous system with a brain exists. This position is self-refuting because it disparages the very entity (mind) required to make the argument. Behe says the mind is real but denied by many academics.

Science has failed to demonstrate that conscious beings could be the result of the random mutation/natural selection mechanism. So, if a mutation/selection process did not build the human brain, what did?

How can we know that minds other than our own exist? Behe answers: from the effects they have. What evidence suggests the operation of a mind? Behe says the evidence for the operation of a mind is a *purposeful arrangement of parts*. The parts could be anything. A mind need not be seen to be detected. The arrangement of chemical parts in living things indicates an intelligence. Richard Dawkins said that biology is the study of things that look like they were designed for a purpose but were really created by the random mutation/natural selection process. But we

now know that the random mutation/natural selection process is inadequate to promote macroevolution. Life was the product of a mind. Behe says that intelligent design in biology is obvious. Science can't be separated from purpose.

Behe says that in order to do science, one must assume the following: reason and logic are valid, the external world exists independent of our thoughts, other minds exist and can be detected by the purposeful arrangement of parts, and we have a functional mind able to detect and comprehend reality. Darwin wondered if a mind generated by evolution would be trustworthy to know the truth. There are two alternatives: affirm materialism and deny your mind or affirm your mind and deny materialism. If the mind is in doubt, then so is reason and any hope of doing science.

People are faced with a choice between intelligent design or the multiverse.[6] If the multiverse is considered, one would expect brains with false ideas to appear.[6] Materialists, by abandoning the mind, embrace irrationality. Some scientists have embraced the idea of a "brain in a vat" reducing all of one's experience to a mere simulation. So, reality is not real, and science is only investigating a simulation. One can't embrace reason yet say the world does not really exist; in so doing, one loses connection with reality. Behe says the denial of the reality of mind is a calamity for science and society.[7]

Evolutionary psychology holds that brains were built for survival and not to understand truth. The brain is only a programmed machine. But materialism is built on Darwinism, and Darwinism has been disproven in the last twenty years. If materialism fails, then so does evolutionary psychology, which holds we are merely the product of heredity and the environment.

How does a nonphysical mind and a physical body and brain interact? We may never be able to fully explain the mind, but that is no reason to reject the faculty with which we understand reality in the first place. Our minds can't be reduced to mere physical processes.

Nature displays the hallmarks of intelligent design.

COMING EVENTS

Thursday, March 12, 7:00 pm, Providence Baptist Church, 6339 Glenwood Ave., Raleigh, Room 237

Creation Versus Cartoon Evolution —and the Winner Is...

Parents and children ten and above: This entertaining and informative talk by our new speaker, Christian businessman and TASC supporter, Xan Spencer, is especially for you and your children to gain and hold faith in this trying world!

From school age all the way through adulthood we are constantly hit with so many evolution "facts" and examples that our mind cannot help but subconsciously take it in. It's so invasive—even our children's cartoons and YouTube shows talk about it often. The question is are we actively educating ourselves and teaching our children the truth so that when they hear or see these things, we can immediately dismiss them as being totally false, just like we immediately dismiss cartoons as not being real—nothing more than made-up characters and stories. Today I'm excited to reignite your faith and share Biblical truths and real-life examples that provide ourselves and children with much needed assurance that this world and everything in it was created by our heavenly Father!

Hello! My name is Xan Spencer, I grew up in Mississippi and attended the University of Southern Mississippi and Middle Tennessee State University, where I majored in business and entrepreneurship. My wife Jenn and I have been married for twelve years, and we have two children. Our son Cullen is seven, and our daughter Maryn is almost three. We have also been active foster parents for over three years. The last twelve years I've worked for myself, starting multiple businesses, of which two have been sold. I've been featured on MSN and MSN Money, among other publications. Most importantly I love the Lord and for the last several years have studied creation science to strengthen my faith, to share His Word with confidence, and to give myself the education necessary to equip my children with truth and confidence in His Word and creation. I look forward to meeting you on March 12 at the TASC meeting!

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[6] For a discussion of the multiverse and the fine-tuning of physics see Reynolds DW (2019) Book review: *A Fortunate Universe: Life in a Finely Tuned Cosmos*. *TASC Newsletter*, October <https://tasc-creationscience.org/article/book-review-fortunate-universelife-finely-tuned-cosmos-part-1> and November <https://tasc-creationscience.org/article/book-review-fortunate-universe-life-finely-tuned-cosmos-part-2>. Accessed 2020 Jan 28.

[7] For a discussion of various worldviews, including materialism, from a Christian perspective see Reynolds DW (2019 August) A review of *Finding Truth* by Nancy R Pearcey. *TASC Newsletter*, <https://tasc-creationscience.org/article/review-finding-truth-nancy-r-pearcey>. Accessed 2020 Jan 28.

So come on out at 7 pm for a great witness for you and your children, and you can still get your children back home and in bed before 9 pm with good edification and great hope for their tomorrow!