
BIBLIOGRAPHIC AND ANNOTATED LIST OF PEER-REVIEWED PUBLICATIONS SUPPORTING INTELLIGENT DESIGN

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PART I: INTRODUCTION

While intelligent design (ID) research is a new scientific field, recent years have been a period of encouraging growth, producing a strong record of peer-reviewed scientific publications.

In 2011, the ID movement counted its 50th peer-reviewed scientific paper and new publications continue to appear. As of 2015, the peer-reviewed scientific publication count had reached 90. Many of these papers are recent, published since 2004, when Discovery Institute senior fellow Stephen Meyer published a groundbreaking paper advocating ID in the journal *Proceedings of the Biological Society of Washington*. There are multiple hubs of ID-related research.

Biologic Institute, led by molecular biologist Doug Axe, is “developing and testing the scientific case for intelligent design in biology.” Biologic conducts laboratory and theoretical research on the origin and role of information in biology, the fine-tuning of the universe for life, and methods of detecting design in nature.

Another ID research group is the Evolutionary Informatics Lab, founded by senior Discovery Institute fellow William Dembski along with Robert Marks, Distinguished Professor of Electrical and Computer Engineering at Baylor University. Their lab has attracted graduate-student researchers and published multiple peer-reviewed articles in technical science and engineering journals showing that computer programming “points to the need for an ultimate information source qua intelligent designer.”

Other pro-ID scientists around the world are publishing peer-reviewed pro-ID scientific papers. These include biologist Ralph Seelke at the University of Wisconsin Superior, Wolf-Ekkehard Lönning who recently retired from the Max Planck Institute for Plant Breeding Research in Germany, and Lehigh University biochemist Michael Behe.

These and other labs and researchers have published their work in a variety of appropriate technical venues, including peer-reviewed scientific journals, peer-reviewed scientific books (some published by mainstream university presses), trade-press books, peer-edited scientific anthologies, peer-edited scientific conference proceedings and peer-reviewed philosophy of science journals and books. These papers have appeared in scientific journals such as *Protein Science*, *Journal of Molecular Biology*, *Theoretical Biology and Medical Modelling*, *Journal of Advanced Computational Intelligence and Intelligent Informatics*, *Complexity*, *Quarterly Review of Biology*, *Cell Biology International*, *Physics Essays*, *Rivista di Biologia / Biology Forum*, *Physics of Life Reviews*, *Quarterly Review of Biology*, *Journal of Bacteriology*, *Annual Review of Genetics*, and many others. At the same time, pro-ID scientists have presented their research at conferences worldwide in fields such as genetics, biochemistry, engineering, and computer science.

Collectively, this body of research is converging on a consensus: complex biological features cannot arise by unguided Darwinian mechanisms, but require an intelligent cause.

Despite ID's publication record, we note parenthetically that [recognition in peer-reviewed literature is not an absolute requirement to demonstrate an idea's scientific merit](#). Darwin's own theory of evolution was first published in a book for a general and scientific audience -- his *Origin of Species* -- not in a peer-reviewed paper. Nonetheless, ID's peer-reviewed publication record shows that it deserves -- and is receiving -- serious consideration by the scientific community.

The purpose of ID's budding research program is thus to engage open-minded scientists and thoughtful laypersons with credible, persuasive, peer-reviewed, empirical data supporting intelligent design. And this is happening. ID has already gained the kind of scientific recognition you would expect from a young (and vastly underfunded) but promising scientific field. The scientific progress of ID has won the serious attention of skeptics in the scientific community, who engage in scientific debate with ID and attend private scientific conferences allowing off-the-record discussion with ID proponents.

In the Table of Contents below, we provide a bibliographic list of the peer-reviewed papers. Following that is an extensive annotated bibliography of technical publications of various kinds that support, develop or apply the theory of intelligent design. The articles are grouped into three categories, according to the type of publication.

PART II: TABLE OF CONTENTS & BIBLIOGRAPHIC LIST OF PAPERS

| | |
|---|----|
| PART I: INTRODUCTION | 1 |
| PART II: TABLE OF CONTENTS & BIBLIOGRAPHIC LIST OF PAPERS | 3 |
| PART III: ANNOTATED BIBLIOGRAPHY OF PEER-REVIEWED PUBLICATIONS | 13 |
| Category 1: Scientific Publications Supportive of Intelligent Design Published in Peer-Reviewed Scientific Journals, Conference Proceedings, or Academic Anthologies..... | 13 |
| Selected Publications from this Category | 13 |
| Annotated Bibliography of Publications in this Category | 15 |
| • Wolf-Ekkehard Lönnig, “Mendel’s Paper on the Laws of Heredity (1866): Solving the Enigma of the Most Famous ‘Sleeping Beauty’ in Science,” <i>eLS</i> (Jon Wiley & Sons, 2017)..... | 15 |
| • Granville Sewell, “On ‘compensating’ entropy decreases,” <i>Physics Essays</i> , Vol. 30:1 (2017). (PDF) | 16 |
| • Ola Hössjer, Ann Gauger, and Colin Reeves, “Genetic Modeling of Human History Part 2: A Unique Origin Algorithm,” <i>BIO-Complexity</i> , Vol. 2016 (4)..... | 18 |
| • Ola Hössjer, Ann Gauger, and Colin Reeves, “Genetic Modeling of Human History Part 1: Comparison of Common Descent and Unique Origin Approaches,” <i>BIO-Complexity</i> , Vol. 2016 (3)..... | 18 |
| • George D. Montañez, “Detecting Intelligence: The Turing Test and Other Design Detection Methodologies,” <i>8th International Conference on Agents and Artificial Intelligence (ICAART 2016)</i> , pp. 517-523 (2016)..... | 20 |
| • Scott T. Matuscak and Change Laura Tan, “Who are the parents of <i>Mycoplasma mycoides</i> JCVI-syn1.0?,” <i>BIO-Complexity</i> , Vol. 2016 (2)..... | 21 |
| • Dustin J. Van Hofwegen, Carolyn J. Hovde, and Scott A. Minnich, “Rapid Evolution of Citrate Utilization by <i>Escherichia coli</i> by Direct Selection Requires citT and dctA,” <i>Journal of Bacteriology</i> , Vol. 198 (7): 1022-1034 (April, 2016)..... | 21 |
| • Douglas D. Axe and Winston Ewert, “ <i>Stylus</i> Experiments Made Easy—A Free App for Personal Computers,” <i>BIO-Complexity</i> , Vol. 2016 (1)..... | 23 |
| • Douglas D. Axe and Ann K. Gauger, “Model and Laboratory Demonstrations That Evolutionary Optimization Works Well Only If Preceded by Invention—Selection Itself Is Not Inventive,” <i>BIO-Complexity</i> , Vol. 2015 (2)..... | 24 |
| • Winston Ewert, “Overabundant mutations help potentiate evolution: The effect of biologically realistic mutation rates on computer models of evolution,” <i>BIO-Complexity</i> , Vol. 2015 (1). ... | 25 |
| • Bhakti Niskama Shanta, “Life and consciousness - The Vedantic view,” <i>Communicative & Integrative Biology</i> , Vol. 8(5): e1085138 (2015). | 26 |

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- David Snoke, “Systems Biology as a Research Program for Intelligent Design,” *BIO-Complexity*, Vol. 2014 (3). 37
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- Granville Sewell, “Entropy and Evolution,” *BIO-Complexity*, Vol. 2013 (2). 44

- Michael J. Denton, “The Place of Life and Man in Nature: Defending the Anthropocentric Thesis,” *BIO-Complexity*, Vol. 2013 (1)..... 45
- Berkley E. Gryder, Chase W. Nelson, and Samuel S. Shepard, “Biosemiotic Entropy of the Genome: Mutations and Epigenetic Imbalances Resulting in Cancer,” *Entropy*, 15: 234-261 (2013)..... 46
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- Selected Publications from this Category 91
- Annotated Bibliography of Publications in this Category 92
- William A. Dembski, Winston Ewert, and Robert J. Marks II, “A General Theory of Information Cost Incurred by Successful Search,” pp. 26- 63, in Robert J. Marks II, Michael J. Behe, William A. Dembski, Bruce L. Gordon, and John C. Sanford eds., *Biological Information: New Perspectives* (Singapore: World Scientific, 2013). 92
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93
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- Jonathan Wells, “Not Junk After All: Non-Protein-Coding DNA Carries Extensive Biological Information,” pp. 210-231, in Robert J. Marks II, Michael J. Behe, William A. Dembski, Bruce L. Gordon, and John C. Sanford eds., *Biological Information: New Perspectives* (Singapore: World Scientific, 2013). 96
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 - Stephen C. Meyer, “The Cambrian Information Explosion: Evidence for Intelligent Design,” pp. 371-391, in William A. Dembski and Michael Ruse, eds., *Debating Design: From Darwin to DNA* (Cambridge, United Kingdom: Cambridge University Press, 2004). 104

- Granville Sewell, "A Mathematician's View of Evolution," *The Mathematical Intelligencer*, Vol. 22(4) (2000) (HTML). 105

Category 3: Articles Supportive of Intelligent Design Published in Peer-Reviewed Philosophy Journals, or Peer-Reviewed Philosophy Books Supportive of Intelligent Design 106

- Selected Publications from this Category 106
- Partially Annotated Bibliography of Publications in this Category 106
- Michael C. Rea, *World without Design: The Ontological Consequences of Naturalism* (Oxford University Press, 2004). 106
- William Lane Craig, "Design and the Anthropic Fine-Tuning of the Universe," in *God and Design: The Teleological Argument and Modern Science*, pp. 155-177. (Neil Manson ed., London: Routledge, 2003). 106
- Michael Behe, "Reply to my Critic: A Response to Reviews of *Darwin's Black Box: The Biochemical Challenge to Evolution*," *Biology and Philosophy*, Vol. 16, 685–709, (2001). 106
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- William Lane Craig, "The Anthropic Principle," in *The History of Science and Religion in the Western Tradition: An Encyclopedia*, pp. 366-368 (Gary B. Ferngren, general ed., Garland Publishing, 2000). 106
- Michael Behe, "Self-Organization and Irreducibly Complex Systems: A Reply to Shanks and Joplin," *Philosophy of Biology*, Vol. 67(1):155-162 (March, 2000). 106
- William Lane Craig, "Barrow and Tipler on the Anthropic Principle vs. Divine Design," *British Journal for the Philosophy of Science*, Vol. 38: 389-395 (1988). 106
- William Lane Craig, "God, Creation, and Mr. Davies," *British Journal for the Philosophy of Science*, Vol. 37: 168-175 (1986). 107

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- Michael J. Behe, “Experimental Evolution, Loss-of-Function Mutations, and ‘The First Rule of Adaptive Evolution,’” *The Quarterly Review of Biology*, Vol. 85(4):1-27 (December 2010).
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- Ann K. Gauger and Douglas D. Axe, “The Evolutionary Accessibility of New Enzyme Functions: A Case Study from the Biotin Pathway,” *BIO-Complexity*, Vol. 2011(1).
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- Dustin J. Van Hofwegen, Carolyn J. Hovde, and Scott A. Minnich, “Rapid Evolution of Citrate Utilization by *Escherichia coli* by Direct Selection Requires *citT* and *dctA*,” *Journal of Bacteriology*, Vol. 198 (7): 1022-1034 (April, 2016).
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- Douglas D. Axe, “Extreme Functional Sensitivity to Conservative Amino Acid Changes on Enzyme Exteriors,” *Journal of Molecular Biology*, Vol. 301:585-595 (2000).
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Annotated Bibliography of Publications in this Category

- **Wolf-Ekkehard Lönnig, “Mendel’s Paper on the Laws of Heredity (1866): Solving the Enigma of the Most Famous ‘Sleeping Beauty’ in Science,” *eLS* (Jon Wiley & Sons, 2017).**

In this peer-reviewed paper, geneticist Wolf-Ekkehard Lönnig asks why Gregor Mendel's accurate theories of heredity developed in the 19th century were initially rejected or ignored by many other scientists. He concludes that it is because at that time, the scientific community was completely enamored with Darwinian evolution and unwilling to consider ideas that did not fit with Darwin's models of evolution and inheritance. As Lönnig puts it:

His [Mendel’s] analysis, discernment and exposition of the laws of heredity as well as his views on evolution diametrically defied and contradicted the ideas and convictions of Darwin and his followers. ... [T]he basic reason for the neglect of the laws of heredity was essentially this: To imply something like a static definition of the species by constant hereditary elements right into a momentous process vigorously favouring the Darwinian revolution (continuous evolution by natural selection without any teleology intimately combined with the inheritance of acquired characteristics, to underscore the latter, often forgotten point once more) was met – although usually silently – with skepticism, deliberate ignorance and strong opposition. And there is no doubt concerning Darwin’s overwhelming victory in the battle for the scientific minds in the nineteenth century, so much so that Mendel’s performance before the

Natural History Society of Brünn was even met with ‘scornful laughter’....

Lönnig quotes Italian biologist Guiseppe Sermonti who concurs with this explanation: “What really happened was that Mendel ruled out almost all the forces that Darwin had invoked to explain evolution.”

Because Mendel’s theory of inheritance produces “all-or-nothing traits,” Lönnig explains that this conflicted with Darwin’s ideas about gradual evolution:

[P]erhaps even more important, Mendel’s discoveries cast doubt on another definitely decisive and essential part of Darwin’s theory: continuous evolution, for which Darwin had postulated ‘infinitesimally small inherited variations’, ‘steps not greater than those separating fine varieties’ and ‘insensibly fine steps’, ‘for natural selection can act only by taking advantage of slight successive variations; she can never take a leap, but must advance by the shortest and slowest steps’.

According to Lönnig, “in Mendel’s view, endless evolution was neither probable for cultivated plants nor for species in the wild.”

- **Granville Sewell, “On ‘compensating’ entropy decreases,” *Physics Essays*, Vol. 30:1 (2017). ([PDF](#))**

In 2011, mathematician Granville Sewell was disallowed from publishing an article in the journal *Applied Mathematics Letters (AML)* simply because it was critical of Darwinian evolution. Then, that non-published paper was later critiqued in the journal *Mathematical Intelligencer*, even though it was never published in the first place. Even worse, his critics doubled-down on censorship by disallowed him from publishing a rebuttal within that journal. Obviously his arguments must hold merit if technical journals are willing devote space to rebutting them. This paper in the journal *Physics Essays* grants Sewell the space to respond to his critics.

Defenders of Darwinian evolution sometimes argue that decreases in entropy in living organisms due to evolutionary processes do not violate the second law of thermodynamics if those decreases are “compensated for” by increases of entropy elsewhere in the universe. Sewell rejoins that “there is no such total entropy, and that the compensation argument is not a valid way to dismiss the claim that evolution violates the second law.” To appreciate the absurdity of the compensation argument, Sewell proposes an imaginary scenario where “a tornado turns a town into rubble, then a second tornado turns this rubble back into houses and cars.” Of course this is wildly improbable, but if the town is an open system, and total entropy of the universe still increases, then according to the compensation argument “the second tornado does not pose any conflict with the second law.” Sewell documents that, unfortunately, various physics textbooks make precisely this sort of mistake. He continues:

[T]o argue that evolution does not violate the second law, you cannot simply dismiss the problem by saying, the Earth is an open system so any decreases in entropy here are easily compensated by increases elsewhere, you have to argue that thanks to the influx of solar energy, it is not really impossibly improbable that the four fundamental, unintelligent forces of physics alone could rearrange the fundamental particles of physics into computers, science libraries, airplanes, and iPhones. Common sense tells us that the fact that order can increase in an open system does not mean that tornados can turn rubble into houses and cars, or that computers can appear on a barren planet as long as the planet receives solar energy. Something must be entering the open system which makes the appearance of computers not extremely improbable, for example, computers.

Sewell then turns to the improper handling of his 2011 *AML* paper, noting that “the reason my accepted *AML* paper was withdrawn was because it seemed to support intelligent design (ID) theory,” even though article “did not explicitly promote intelligent design.” He then recounts the conclusion of that paper:

Of course, one can still argue that the spectacular increase in order seen on Earth does not violate the second law because what has happened here is not really extremely improbable. And perhaps it only seems extremely improbable, but really is not, that, under the right conditions, the influx of stellar energy into a planet could cause atoms to rearrange themselves into nuclear power plants and spaceships and digital computers. But one would think that at least this would be considered an open question, and those who argue that it really is extremely improbable, and thus contrary to the basic principle underlying the second law of thermodynamics, would be given a measure of respect, and taken seriously by their colleagues, but we are not.

In this paper, he offers a similar conclusion:

If Darwin was right, then evolution does not violate the second law because, thanks to natural selection of random mutations, and to the influx of stellar energy, it is not really impossibly improbable that advanced civilizations could spontaneously develop on barren, Earth-like planets. Getting rid of the compensation argument would not change that; what it might change is, maybe science journals and physics texts will no longer say, sure, evolution is astronomically improbable, but there is no conflict with the second law because the Earth is an open system, and things are happening elsewhere which, if reversed, would be even more improbable.

The publication of this peer-reviewed paper by Sewell might be evidence that physics texts will one day move in the right direction.

- **Ola Hössjer, Ann Gauger, and Colin Reeves, “Genetic Modeling of Human History Part 2: A Unique Origin Algorithm,” *BIO-Complexity*, Vol. 2016 (4).**

In their prior paper, these authors laid out a framework for testing models of human origins where humans share a common ancestor with chimpanzees, and also models where humans experienced a “unique origin” and were “created with considerable diversity.” This second paper presents mathematical algorithms “for testing different historical scenarios of the human population,” including common ancestry models, and models where humans “all descend from one single couple.” Their mathematical approach can simulate human history by varying different parameters, including population expansion, bottlenecks, colonization and migration patterns, mating and reproduction schemes, and various types of mutations in autosomal chromosomes, sex chromosomes, and mitochondrial DNA. Additionally, “[a]n important parameter of the model is the created diversity of the founder generation, since it facilitates a higher degree of genetic diversity for a relatively young population within autosomal and X chromosomal regions, and possibly also for mitochondrial DNA.”

Their algorithms incorporate what they identify as the six major mechanisms of genetic change, (i) genetic drift, (ii) genetic recombination, (iii) colonization and migration, (iv) mutations, (v) natural selection, and (vi) initial created founder diversity. They note that “common descent models only include the first five mechanisms, but (vi) is important in order to generate enough diversity for a population with only one founding couple.” Indeed, they observe that a “particularly important parameter is the created diversity, which makes it possible to obtain a substantial amount of genetic diversity for nuclear autosomal and X-chromosome DNA, during a relatively short period of time.”

After going through a detailed mathematical analysis of the model they conclude, “[i]n subsequent papers, we plan to simulate human DNA data from our proposed model in order to assess how well it fits real data,” with the ultimate goal of finding “the best fitting population history within a unique origin framework, and then to compare it with a best fitting common ancestry model.”

- **Ola Hössjer, Ann Gauger, and Colin Reeves, “Genetic Modeling of Human History Part 1: Comparison of Common Descent and Unique Origin Approaches,” *BIO-Complexity*, Vol. 2016 (3).**

Did humans evolve from apelike creatures or were they intelligently designed? According to the standard evolutionary view, humans share a common ancestor with chimpanzees, and our lineage diverged about 6 million years ago in Africa and then evolved by unguided evolutionary mechanisms into its present form. This paper evaluates the assumptions underlying the standard evolutionary model of human origins and finds “it is full of gaps and weaknesses.” Instead, the authors maintain that “a unique origin model where humanity arose from one single couple with created diversity seems to explain data at least as well, if not better.”

After reviewing five main mechanisms invoked by standard evolutionary models of population genetics to explain human genetic diversity (mutation, genetic drift, natural selection, recombination, and colonization and migration), the paper observes that:

Neo-Darwinism accounts for the above-mentioned mechanisms I–V, and among them germline mutations are essentially the only way by which novel DNA can arise. The theory does not allow for large amounts of new and suddenly appearing diversity. The reason is that neo-Darwinism is framed within methodological naturalism. This prevailing approach to science only allows for natural hypotheses. But if an intelligent designer is invoked as a possible explanation, and if humanity originates from one single couple, it is possible that their chromosomes were created with considerable diversity from the beginning.

Thus, the authors report discovery of “a sixth mechanism of genetic change,” one which is almost universally ignored by evolutionary models: “Created founder diversity is biologically plausible for DNA of non-sex chromosomes.”

With these mechanisms in mind, the article compares standard evolutionary “common ancestry” models of human origins with “unique origin” models, where an initial pair of humans was created with significant founder diversity. There are two main common ancestry models of human origins: the Out-of-Africa model, where humans evolved in Africa and then migrated out one single time, and the Multiregional Evolution model, where humans evolved in Africa but migrated out multiple times, with different human populations around the world evolving in parallel. There are also two “unique origin” models: An African Ancestry model, where the initial created pair was located in Africa, and a Middle East ancestry model, where an initial couple was created in the Middle East and then humans migrated around the world.

The authors note that the “main argument against a unique origin is that the nucleotide diversity of human DNA data seems too high in order make a single founding couple possible.” But they argue it is possible that humans are descended from an initial couple if “they were created with genetic diversity in their autosomal and X-chromosome DNA.” What about the location of the initial couple? Non-African populations of humans seem more genetically similar compared to African humans, and they note that “the Middle East ancestry model faces some challenges, in particular to explain why African DNA looks older than non-African DNA.” However, a Middle East origin model could explain the data if “the age of humanity is much more recent” than common ancestry models predict, and if African populations experienced higher rates of genetic change and lived in isolated communities where unique diversity was easily fixed into small populations. They cite previous literature to support these ideas, making the Middle East unique origin model a realistic possibility.

The authors conclude that “Any common descent model faces a challenge to explain the genetic differences rather than the similarities with other species, the consequences of

inbreeding depression and increased genetic entropy, human DNA mixture with archaic populations, and that our DNA resembles a mosaic of about four founder genomes.” Thus, they find that “The provisional conclusion is that a unique origin model seems more plausible.” But which unique origin model best explains the data? They urge future research is needed to test the two unique origins models, which is what the authors plan to do in subsequent papers. It may be that multiple models can explain the data, in which case they conclude that “the common descent model of our origin from ape-like ancestors can no longer be claimed as conclusive proof that there could not have been a single first pair.”

- **George D. Montañez, “Detecting Intelligence: The Turing Test and Other Design Detection Methodologies,” *8th International Conference on Agents and Artificial Intelligence (ICAART 2016)*, pp. 517-523 (2016).**

The ‘Turing Test’ is a famous test proposed by computer scientist Alan Turing for determining whether a computer had achieved artificial intelligence. In this peer-reviewed paper, computer scientist George D. Montañez proposes that the Turing Test basically amounts to a form of design detection—an attempt to determine if an intelligent agent is at work. Montañez explains that if we accept the Turing Test as valid, then we must consider the possibility of intelligent design:

[A] simple argument establishes the equivalence of the Turing Test to intelligent design methodology in several fundamental respects. Constructed with similar goals, shared assumptions and identical observational models, both projects attempt to detect intelligent agents through the examination of generated artifacts of uncertain origin. Second, if the Turing Test rests on scientifically defensible assumptions then design inferences become possible and cannot, in general, be wholly unscientific. Third, if passing the Turing Test reliably indicates intelligence, this implies the likely existence of a designing intelligence in nature. ... For the Turing Test to work, one must be able to distinguish intelligent causes from unintelligent causes based solely on observable artifacts. But this leads to the conclusion that intelligent design cannot be simultaneously disregarded, since its methodological structure rests on the same foundation. Furthermore, if the Turing Test is a reliable procedure for detecting intelligence, then the cause of biological origins is likely an intelligent mind, having passed a generalized Turing Test...

Montañez notes that critics of the Turing Test argue that we cannot detect artificial intelligence. But he rejoins that if ID is correct that intelligent agency can be detected, then this lends credence to the Turing Test. His conclusion takes aim at claims that we are never allowed to question the consensus: “To allow for the possibility of intelligent design would be to deny a scientific consensus, and we are often reminded that true scientists and intelligent laymen cannot deny any fact established by the consensus of experts. To do so is derided as a strong form of denialism, for suggesting the majority of scientists might be wrong on a well-studied issue. Surely such could not be the case.”

- **Scott T. Matuscak and Change Laura Tan, “Who are the parents of *Mycoplasma mycoides* JCVI-syn1.0?,” *BIO-Complexity*, Vol. 2016 (2).**

In 2010, the noted biotechnologist J. Craig Venter seemingly bolstered the idea that blind mechanistic processes could create life when his team announced the creation of “the first self-replicating species we’ve had on the planet whose parent is a computer.” This peer-reviewed paper scrutinizes that research and finds that in actuality, the true parents of the bacterium *M. mycoides* were previous bacteria of the same species, with small amounts of input from yeast and *E. coli*, as well as the ingenuity of humans. After analyzing the methods used in the research, they find that “the computer was only used, passively, to store genome sequence information. It did not generate a single molecule necessary for the survival or arrival of” the bacterial cells. According to their analysis, the vast majority of the bacteria’s genome was based upon DNA from the genomes of living bacteria. To be precise:

[T]he final complete genome, 98.55% of the genome sequence was based on the natural *M. mycoides* genome sequence, 0.94% was the yeast cloning vector sequence, and 0.08% came from bacterial insertions. The last 0.43% was designed by humans in the form of watermarks, using a computer as a tool to convert the letters, numbers, and punctuation into DNA sequences.

They further observe that “the human-engineered watermark sequences do not produce any functional products within the cell, so even the small percentage of sequences that were actually designed by humans using computers do not affect the cell with respect to function (except perhaps as a burden to maintain those sequences.”) Thus, they argue that “if one were to classify the parents of an organism on the basis of the providers of the genetic sequence, then we should consider the parent of *M. mycoides* JCVI-syn1.0 to be *M. mycoides* because it provides almost 99% of the genome sequence.” They conclude that “regardless of which criteria one chooses to use in order to define what constitutes the actual parent for the *M. mycoides* JCVI-syn1.0 cells, the computer would be the least plausible candidate. It was just a place that was used by humans to store the sequences in transit. The sequence on a computer will not give birth to even a single DNA, RNA, or protein molecule of any cell.” In Venter’s research, no blind mechanisms created any new species.

- **Dustin J. Van Hofwegen, Carolyn J. Hovde, and Scott A. Minnich, “Rapid Evolution of Citrate Utilization by *Escherichia coli* by Direct Selection Requires *citT* and *dctA*,” *Journal of Bacteriology*, Vol. 198 (7): 1022-1034 (April, 2016).**

For years, Richard Lenski’s Long Term Evolution Experiment (LTEE) has been touted as showing that *E. coli* bacteria evolved a “new” complex trait—the ability to uptake and metabolize the molecule citrate. The LTEE required 30,000 generations and many years to acquire the supposedly “new” trait. But this peer-reviewed research study, co-authored by biologist Scott Minnich, witnessed the same trait arise in only about 12 generations and 30 days. This suggests that this trait is not very genetically complicated,

and that there is more to the story than has been told. Indeed, this paper shows that no new genetic information arose during the evolution of this trait.

LTEE proponents often fail to acknowledge that *E. coli* normally have the ability to feed on citrate—the bacteria just cannot uptake and metabolize citrate under oxic conditions. In the LTEE, bacteria evolved the ability to uptake citrate under oxic conditions (the “Cit+ phenotype”). But did anything new evolve? At the genetic level, Minnich and his coauthors’ research says the answer is “no.” To understand why, review the three primary mutations required to produce the Cit+ phenotype:

- A mutation allowed the *E. coli* to express an antiporter protein, CitT, under oxic conditions. CitT permits one molecule of citrate to be imported into the cell in exchange for one of three less ‘valuable’ molecules with less carbon: succinate, fumarate, or malate. However, gene for this antiporter protein already existed previously, so no new gene evolved.

CitT is usually switched off in *E. coli* when oxygen is present, but this mutation allowed it to be turned on. What caused it to become turned on? Biochemically speaking, a switch that normally represses expression of the gene that produces CitT under oxic conditions was broken via the mutation, so the citrate-uptake pathway got turned on under oxic conditions. This isn’t the evolution of a new molecular feature; it’s the breaking of a molecular feature—a repressor switch.

- There was a duplication mutation of the gene for the CitT antiporter protein, allowing the bacteria to produce more of that protein. This allowed more citrate to be uptaken under oxic conditions. This too does not involved the evolution of anything new—it only involves making more of something already present.
- Another gene duplication mutation occurred for the gene that produces the protein DctA, a succinate importer. This allowed some of the succinate that had been lost in exchange for citrate to be recovered and transported back into the cell. Again, this is just making more of something already present; nothing new arose.

Thus, the mutational pathway observed in the LTEE which generated the Cit+ phenotype involves: (i) Breaking something at the molecular level (a repressor), (ii) Making more of something already present (citrate importer), and (iii) Making more of something already present (succinate importer). Such changes—breaking features at the molecular level, or making more of some pre-existing components—have been long known to be possible under Darwinian evolution. As Minnich and his coauthors explain in their paper: “No new genetic information (novel gene function) evolved.” They also write, “the LTEE has not substantiated evolution in the broader sense by generation of new genetic information, i.e. a gene with a new function.” They conclude:

Finally, because this adaptation did not generate any new genetic information and only required expanded expressions of two existing transporters (citT and dctA), generation of *E. coli* Cit+ phenotypes in our estimation do not warrant consideration as a speciation event.

For microbiologists, however, a key question is why did this paper's research observe the Cit+ phenotype arise so rapidly, whereas Lenski's LTEE required a long time for the same? A commentary in the *Journal of Bacteriology* that accompanied this paper research paper explains:

[T]he primary message of the paper by Van Hofwegen et al. is that the series of events used to explain adaptation in the short-transfer LTEE (and in speciation) might need to be revised. ... It would appear that the delay in the LTEE experiments may not reflect need for a neutral potentiation step, but the difficulty of intermittent selection to act on frequent copy number variants. The bottleneck in serial dilutions is hard to cross when initial improvements are due to an unstable copy number variant that is counter-selected during the intervening rapid growth period. (John Roth and Sophie Maisnier-Patin, "Re-interpreting long-term evolution experiments -- Is delayed adaptation an example of historical contingency or a consequence of intermittent selection," *Journal of Bacteriology*, Vol. 198:1009-1012 (April, 2016).)

The phrase "may not reflect need for a neutral potentiation step" means that no complex sequence of neutral mutations was needed to produce the Cit+ phenotype. Essentially this research shows that when one imposes strong selection for growth on citrate, the story isn't one of neutral evolution evolving a complex feature, but one where each step gave a successive advantage (and no step creates anything genetically new). Under the right selection pressures, this relatively simple phenotype can arise very quickly. This research shows that Lenski's work not the impressive story of a complex evolutionary pathway many claimed it was.

Most importantly, this paper shows that Lenski's work did not demonstrate the evolution of any new biochemical features. Rather, it takes pre-existing transporter proteins and over-expresses them in an unusual environment—but only by breaking a molecular switch. Biochemically, these molecules are only doing what they were already designed to do. Nothing "new" evolved here—and in that sense no new genetic information was produced.

- **Douglas D. Axe and Winston Ewert, "Stylus Experiments Made Easy—A Free App for Personal Computers," *BIO-Complexity*, Vol. 2016 (1).**
Stylus is a computer program that models Darwinian evolution *in silico* by trying to evolve Chinese characters. Because Chinese characters have a structure that is related to their function, testing the evolution of Chinese characters provides a meaningful analogue for testing the evolvability of new proteins. *Stylus* thus models the Darwinian

process in a more biologically realistic manner than many other would-be evolutionary simulations. This peer-reviewed paper describes and announces a free *Stylus* app for use on personal computers (Mac, PC, Linux). As the paper explains, the *Stylus* app allows users to test the evolvability of various genes (Chinese characters) provided in a library included with the program.

Within the *Stylus* app, genes can be subjected to various types of mutations chosen in experiments run by the user. The program also shows mutation statistics, showing “the effects of point mutations on the selected gene.” The degree to which mutations have optimized gene function can be displayed and exported graphically. The goal is that “the major improvement in ease of use [of *Stylus*] brought about by this new app will attract new users among professional researchers, teachers and students,” creating a growing base of users, and allowing more research on protein evolution to take place.

- **Douglas D. Axe and Ann K. Gauger, “Model and Laboratory Demonstrations That Evolutionary Optimization Works Well Only If Preceded by Invention—Selection Itself Is Not Inventive,” *BIO-Complexity*, Vol. 2015 (2).**

This paper reports original experimental and theoretical research which challenges the evolvability of new protein functions. In a previous paper (“Enzyme Families--Shared Evolutionary History or Shared Design? A Study of the GABA-Aminotransferase Family”), the authors tried to experimentally convert proteins to perform the functions of other closely related proteins, and showed that such an enzyme conversion would require more mutations than would be feasible over the history of life. Darwinian evolutionists often invoke the “promiscuity hypothesis,” wherein a protein has a primary function but might also have some side activity with a weakly selectable function. In time, that side-activity might be refined and optimized to perform some new function very well, and the original primary function may even be lost. In the previous paper, the authors used starting proteins with functions very different from the functions of the target proteins, which precluded promiscuity from successfully aiding the evolution of the new target protein function. This paper, however, tested the promiscuity hypothesis through both experimental lab-work and theoretical simulations.

Experimentally, this study began with a “junk” protein with weak activity against the antibiotic ampicillin, but without a properly folded enzymatic structure for that function. It could not be improved by three rounds of random mutation and selection. In contrast, a weakly functional protein with a destabilized but properly folded structure could rapidly be optimized to wild-type levels of activity. This suggests that the promiscuity hypothesis only works if a protein already has the right kind of functional protein fold. Without that, protein promiscuity cannot lead to the evolution of a new feature.

The authors tested this same question through a theoretical study using *Stylus*, a computer model co-developed by Douglas Axe that simulates Darwinian evolution in a biologically realistic manner by evolving Chinese characters. They started with a random sequence whose product had very weak similarity to the target character, and then

sought to evolve the target through random mutation and selection. This was unsuccessful. Next, they tested whether an already-existing character with some weak similarity to the target could be evolved by mutation and selection to a proficient version of the target character. Once again, this could not be done. However, they found that if the starting character was only six mutations away from optimization, it improved rapidly upon mutation and selection.

This paper thus presents both experimental and theoretical research that converge on a common conclusion: selection and mutation can refine things that already have a well-honed function, in particular where the starting protein already exists as a functional fold of the right design. But if the starting point isn't already near the final target, then unguided evolutionary mechanisms cannot generate new protein folds or novel functions.

- **Winston Ewert, "Overabundant mutations help potentiate evolution: The effect of biologically realistic mutation rates on computer models of evolution," *BIO-Complexity*, Vol. 2015 (1).**

Computer simulations of evolution are often cited as demonstrating the efficacy of the Darwinian mechanism to create new complex features. But do these computer models accurately represent biological reality? In this peer-reviewed paper, computer scientist Winston Ewert analyzes computer models of evolution and finds that they use unrealistically high mutation rates which allow the artificial simulations to evolve new features much more easily than would be feasible in real biological systems. For example, Ewert finds that the programs *Avida* and *Ev* use "a substitution mutation rate of 0.0025 per instruction" and "a substitution rate of approximately 0.0038 per nucleotide," respectively. Yet in the real world, "viruses have mutation rates ranging from 10^{-4} to 10^{-8} per base pair per generation" and "higher organisms sho[w] mutation rates ranging from 10^{-7} to 10^{-11} ." According to Ewert, "the most rapidly mutating viruses undergo mutations at a rate an order of magnitude less than these computer models." He thus finds:

The computer models developed thus far do not solve simple problems when using a biologically realistic mutation rate. ... [W]hen using realistic mutation rates, these models no longer function effectively. This undermines the argument that they support Darwinian evolution and raises a serious challenge to claims of the effectiveness of Darwinian evolution in solving real-world biological challenges.

In particular, Ewert finds that the overestimation of real-world mutation rates within evolutionary simulations creates severe problems for Darwinism when the evolution of a feature requires potentiating mutations—mutations that have no effect when they initially occur, but are required later in the pathway to allow some function to emerge. Ewert explains:

The models fail due to the necessity and difficulty of obtaining potentiating mutations. That is, in each of the models, it is impossible to evolve the solution one beneficial mutation at a time. Some of the mutations necessary to solve the problem will be neutral or deleterious when they first arise. These are called potentiating mutations because they are not helpful by themselves, but introduce the potential for other mutations to be beneficial.

Using computer simulations with biologically realistic parameters, Ewert finds that biologically realistic mutation rates are simply too low to generate needed potentiating mutations. He concludes: “We have argued based on computer models and biological data that [1] potentiating mutations are necessary for adaptation, [2] individual potentiating mutations are very improbable, and [3] there are only a handful available at any point in time. If these three facts are true, there is no way that Darwinism can account for human evolution. For Darwinism to be true, one or more will have to be overturned.”

- **Bhakti Niskama Shanta, “Life and consciousness - The Vedantic view,” *Communicative & Integrative Biology*, Vol. 8(5): e1085138 (2015).**

This peer-reviewed paper in a mainstream biology journal promotes goal-directed, teleological, nonmechanistic, and nonreductionist Vedantic views of biology that are friendly to intelligent design. The article notes that while mainstream science “presumes life as just a chance occurrence,” the Vedantic view holds that “the origin of everything material and nonmaterial is sentient and absolute (unconditioned).” It finds evidence for this viewpoint in the basic biological principle *omne vivum ex vivo*—“life comes from life,” which parallels a Vedantic proverb implying that “An ‘organic whole’ cannot arise from parts that have to be assembled. That process can only produce inorganic, mechanical or chemical processes, not living organisms.” The article then explains that Darwinian evolution cannot explain the “goal-oriented or teleological activities” found in living organisms:

Life’s ability to preserve its own species offers a significant challenge to Darwinian gradualism. Living organisms exhibit many such overtly noticeable goal-oriented or teleological activities (self-determination, self-formation, self-preservation, self-reproduction, self-restitution and so on), which make them distinct from insentient mechanical and chemical systems. Darwin’s *Origin of Species* invokes natural selection to explain the goal-driven activities of the living organisms, but insists that random mutations are exclusively responsible for the gradual but steady appearance of more complicated organisms. This irrational inability to scientifically explain how novel body types arise in study of life and its evolution is the major deficiency of Darwinism.

These problems apply not only to the physical properties of organisms, but also to their behaviors, since “Both abiogenesis and evolution theory are outcomes of mechanistic or reductionistic thinking and that is why they cannot explain how organisms have

cognitive features like thinking, feeling and willing.” According to the article, consciousness is not explainable in mechanistic terms, for “Life and its evolution cannot be understood by imposing simplistic Darwinian mechanistic reductionism on sentient biological systems. Evidence is forcing biologists to go beyond physics and chemistry to properly comprehend the science of consciousness.” While some may argue that these Vedantic views are different from intelligent design, such teleology in biological origins is in fact totally consonant with an intelligent paradigm of biology.

- **Winston Ewert, William A. Dembski, Robert J. Marks II, “Measuring meaningful information in images: algorithmic specified complexity,” *IET Computer Vision*, Vol. 9 (6): 884-894 (December, 2015).**

This peer-reviewed paper applies algorithmic specified complexity (ASC) as a measure of meaning vs. randomness in a dataset, providing a rigorous mathematical analytical tool for detecting design. The authors test this methodology by comparing computer “images which contain content from those which are simply redundancies, meaningless or random noise.” According their design detection model:

For an image to be meaningfully distinguishable, it must relate to some external independent pattern or specification. The image of the sunset is meaningful because the viewer experientially relates it to other sunsets in their experience. Any image containing content rather than random noise fits some contextual pattern. Naturally, any image looks like itself, but the requirement is that the pattern must be independent of the observation and therefore the image cannot be self-referential in establishing meaning. External context is required. If an object is both improbable and specified, we say that it exhibits ‘specified complexity’.

How can we detect whether an image contains specified complexity? They explain that it must be both uncompressible (complex) and match a pattern:

The more the image can be described in terms of a pattern, the more compressible it is, and the more specified. For example, a black square is entirely described by a simple pattern, and a very short computer programme suffices to recreate it. As a result, we conclude that it is highly specified. In contrast, an image of randomly selected pixels cannot be compressed much if at all, and thus we conclude that the image is not specified at all. Images with content such as sunsets take more space to describe than the black square, but are more specified than random noise. Redundancy in some images is evidenced by the ability to approximately restore groups of missing pixels from those remaining.

A simple black square might be compressible and specified, but it is not complex. As they note, “The random image is significantly more complex, whereas the solid square is much less complex.” But these are relatively easy cases. They then try to tackle more difficult images, such as a photograph of the famous scientist Louis Pasteur with

increasing amounts of random noise added. As ASC predicts, the more noise that's added to the image, the lower the ASC. Similarly, resizing an image of Einstein causes it to lose clarity, and its ASC decreases. This is what their model predicts.

What about a picture of “stick men on a sea of noise”? They found that ASC was still able to detect the presence of a complex and specified feature even when surrounded by noise. They conclude that ASC is an effective methodology for distinguishing random image data from meaningful images:

We have estimated the probability of various images by using the number of bits required for the PNG encoding. This allows us to approximate the ASC of the various images. We have shown hundreds of thousands of bits of ASC in various circumstances. Given the bound established on producing high levels of ASC, we conclude that the images containing meaningful information are not simply noise. Additionally, the simplicity of an image such as the solid square also does not exhibit ASC. Thus, we have demonstrated the theoretical applicability of ASC to the problem of distinguishing information from noise and have outlined a methodology where sizes of compressed files can be used to estimate the meaningful information content of images.

The applicability to intelligent design is clear: if ASC is a useful tool for distinguishing designed images from random ones or ones produced by some unguided algorithm, then perhaps it can be applied to biological systems or other natural structures to detect design there as well.

- **David W. Snoke, Jeffrey Cox, and Donald Petcher, “Suboptimality and Complexity in Evolution,” *Complexity*, Vol. 21(1): 322-327 (September/October, 2015).**

This article, by physicists David Snoke, Jeffery Cox, and Donald Petcher, begins by observing that in order to produce a new system, evolution must first try many new variations upon which natural selection can act in order to “find” something useful to retain. But that comes with a potentially fatal cost, since most new variations won't function, leading to the accumulation of “junk.” As the authors put it: “[T]here is an additional energy cost to increased complexity. ... In real systems, building new systems is costly, and the cost of carrying along useless or redundant systems is one of the arguments for the efficiency of existing living systems, as excess baggage is dropped as too costly. The problem can be circumvented by providing an incentive system or reward for trying out new variations. This poses a catch-22 for Darwinian evolution: If the “reward” isn't high enough, nothing new ever evolves. On the other hand, if the reward is too high, too many new things are tried, many of which don't do anything useful, and the system accumulates much deleterious junk. As they explain:

There are two competing processes. On one hand, the energy cost of carrying vestigial systems makes them weakly deleterious, not neutral, which tends to reduce their number. Conversely, without stabs in the dark, that is, new systems

which might eventually obtain new function but as yet have none, no novelty can ever occur, and no increase of complexity. Thus, if the energy cost of vestigial systems is too high, no evolution will occur.

Evolutionary biologists often attempt to resolve this dilemma by claiming it's easy to evolve new structures, but lots of junk accumulates. These authors observe that such reasoning "has historically led evolutionary theorists to expect, that living systems carry a significant fraction of vestigial, or nonfunctional, elements, as well as quasivestigial elements which function with much less than optimal efficiency." To test this evolutionary expectation, they constructed a model that rewards evolving a new function, but that exacts a price for evolving systems that require lots of parts before providing an advantage. They found that when the model was optimized to reward the evolution of new features, it did evolve new features. Some of those features were useful. But the vast majority were not. But there was a cost for the ability to evolve something new. Before the simulation finished, the population experienced a crash because the organisms accumulated so much genetic garbage—new features that were in fact no more than useless freeloaders—that fitness dropped precipitously. Thus, the authors observe another problem: "nature does not reward complexity per se, it rewards functions that enhance survival and reproduction" and "there may be many paths to the same function, some simpler and some more complex, and all will be rewarded roughly the same whether or not the function is done elegantly or not; only the overall energy cost will deter some versions of obtaining the function." Their model tries to accommodate these facts by incorporating "(1) an energy cost for increasing number of elements produced and (2) multiple paths to beneficial functions." There is thus a ratio of reward to the cost of trying out something new:

- If the ratio is too low, then it's costly to try new things, and they will be eliminated right away. New features don't evolve.
- If the ratio is high, then new features will evolve quite easily. Initially, new complexity is generated, and strongly harmful or costly vestigial traits are eliminated. But trying lots of new variation means slightly deleterious traits cannot be weeded out. Over time unhelpful traits accumulate. Eventually such mutations pile up to an extent that the population reaches a crisis point, and crashes. The junk has become an unbearable burden. The organisms go extinct.

According to their simulation, Darwinian evolution thus either produces (1) nothing new, or (2) large amounts of junk that is ultimately deadly. In case (2), the reward for trying new things is high compared to the cost of building new structures. But in order for the ratio to be high enough for complexity to increase, the cost of building new things must be negligible. Novelties proliferate, but the fraction of the beast that's vestigial grows, and the organism is eventually swamped and overwhelmed by harmful vestigial features. However, when trying to avoid the problem of (2) by making the reward-to-cost ratio lower, as in (1), then nothing new ever evolves. Real biological organisms are closer to position (1), because there are efficient ways to get rid of

nonfunctional features that exact a cost and because we don't observe systems that are full of dead weight. But if organisms are in position (1), that suggests new complex features cannot be built because it's very difficult to try new things. They conclude:

In existing living systems, the fitness collapse seen in this model appears to be prevented by mechanisms which quickly eliminate nonfunctional elements, while leaving functional elements untouched. This type of mechanism would seem to prevent 'stabs in the dark' of any great magnitude, and thus prevent ongoing increase of complexity.

When it comes to generating viable living systems, Darwinian evolution faces a 'damned if you do, damned if you don't' dilemma. Whatever cause generated the complex functional biological features we observe, it was not unguided Darwinian evolution.

- **Wolf-Ekkehard Lönnig, "Transposons in Eukaryotes (Part B): Genomic Consequences of Transposition," *eLS [Encyclopedia of Life Sciences]*. John Wiley & Sons, Ltd: Chichester, DOI:10.1002/9780470015902.a0026265 (August, 2015).**

This peer-reviewed article reviews the role of transposable elements (TEs) and, citing to the work of Michael Behe, argues that "irreducibly complex" structures may defy explanation by TEs or standard Darwinian mechanisms:

[M]utation and selection may not be the full explanation for the origin of species; that is, the factors of the neo-Darwinian scenario may find their limits, for example, in the generation of 'irreducibly complex structures' (Behe, 2006, 2007). This is a term used to describe structures that, according to Behe, cannot be explained by a piecemeal production via intermediate steps. Among the examples discussed by Behe are the origins of (1) the cilium, (2) the bacterial flagellum with filament, hook and motor embedded in the membranes and cell wall and (3) the biochemistry of blood clotting in humans. Moreover, the traps of *Utricularia* (Lönnig, 2012) and some other carnivorous plant genera as well as several further apparatus in the animal and plant world appear to pose similar problems for the modern synthesis (joints, echo location, deceptive flowers, the reproductive system of the Australian gastric brooding frog *Rheobatrachus silus*, the mechanical gears of the nymph stage of the leaf hopper *Issus coleoptratus* etc.). Up to now, none of these systems has been satisfactorily explained by neo-Darwinism. Whether accelerated TE activities with all the above named mutagenic consequences can solve the questions posed remains doubtful in the eyes of the critical observer. Moreover, natural selection itself may not have the stringency usually ascribed to it (for details, see ReMine, 1993; Lönnig, 2001, 2012, 2014).

While unguided mutational processes involving TEs seem incapable of producing irreducibly complex structures, the article notes that there may be "teleologic benefits" from TE activities: "Concerning the totally unexpected and extraordinarily high level of

current DNA transposition activities in bats in clear contrast to near extinction or absence of such elements in all other mammals, Huang *et al.* (2012) give sympathetic consideration to 'teleologic benefits' (among others) promoting active DNA transposons in the order Chiroptera (perhaps via HT; Tang *et al.*, 2015). A 'pacemaker proponent' *sensu lato* may perhaps ask whether teleologic benefits could also be involved in an independent origin of the *Transip* TEs and the immune system of jawed vertebrates (not to mention teleology in the sense of Behe, 2006, 2007)."

The article also cites ID authors such as Jonathan Wells and Richard Sternberg while noting that they and other authors think that non-coding DNA is largely functional:

In the wake of the ENCODE (encyclopedia of DNA elements) project, several authors are even favouring positions that almost approach the assumption of 100% functional DNA in all genomes, that is, there is no junk DNA in the genomes of plants and animals at all (Shapiro and von Sternberg, 2005; Wells, 2011).

It concludes by noting that "several lines of evidence" including "irreducibly complex systems" challenge current evolutionary models and should spur us to follow the evidence "wherever it may lead."

- **Mohit Mishra, Utkarsh Chaturvedi, K. K. Shukla, "Heuristic algorithm based on molecules optimizing their geometry in a crystal to solve the problem of integer factorization," *Soft Computing*, DOI 10.1007/s00500-015-1772-8 (July 23, 2015).** This peer-reviewed paper does not argue for intelligent design, but it uses core ID concepts as a heuristic for solving certain kinds of mathematical problems. In that regard, the paper favorably cites and employs the work of ID researchers William Dembski and Robert Marks of the Evolutionary Informatics Laboratory. The paper discusses integer factorization, or how we determine what prime numbers can be multiplied to yield another particular integer. This is essentially a search problem with applications in cryptography and other computer science questions. This is a very difficult problem but some algorithms have been developed to solve it. But which search algorithms are more efficient than others at solving the search? Such a fitness question is precisely what Dembski and Marks address in their research. The paper states:

To quantify the quality of an objective function, we analyze our objective functions based on conservation of information in search theory (Dembski and Marks 2009).

Dembski and Marks have developed a principle of "conservation of information" which holds that if an algorithm does better than blind search, that is because it was given prior information, where the amount of prior information equals at least the measure of how far the algorithm outperforms blind search. Searches can thus perform better than a random search when they are fed information (called "active information") to help

find the target. According to their methodology, Exogenous information (I_{\pm}) represents the difficulty of a search in finding its target with no prior information about the target's location. Active information (I_{+}) is the amount of information smuggled in by intelligence to aid the search algorithm in finding its target. Endogenous Information (I_{\pm}) then measures the difficulty the search will have in finding its target after the addition of Active Information. Thus, $I_{+} = I_{\pm} - I_{\pm}$.

After discussing various methods of solving the problem of integer factorization, this paper asks how the methods work, writing: "In this section, we analyze our objective function based on conservation of information in search (Dembski and Marks 2009)." The authors then cite to Dembski and Marks's concepts like "endogenous information," "exogenous information," and "active information." After discussing how this methodology relates to solving a search question, they conclude, "The conservation of information in search provides a way to quantify the quality of an objective function."

What does all this have to do with Darwinian evolution? The research by Dembski and Marks is applicable to essentially any search function. While this paper focuses on solving the problem of searching for prime numbers that can be multiplied to yield a given integer, Darwinian evolution is, at its heart, also a search algorithm. It uses a trial-and-error process of random mutation and unguided natural selection to find genotypes (i.e., DNA sequences) that lead to phenotypes (i.e., biomolecules and body plans) characterized by high fitness (i.e., fostering survival and reproduction). According to Dembski and Marks, unless you start off with some information indicating where peaks in a fitness landscape may lie, any search—including a Darwinian one—is on average no better than a random search. In some cases, even a random search can work when you have lots of probabilistic resources (i.e., time and opportunities for computation) or when there are lots of targets out there waiting to be found. Thus, Darwinian evolution can work when only one mutation is needed to give some advantage and when evolution takes place within a large, rapidly reproducing population (like we often see in bacteria).

But when targets are rare and there aren't many opportunities for the search (e.g., trying to evolve a complex multimutation feature in long-lived organisms like humans with small effective breeding populations), then such a random search won't work. The paper under discussion here doesn't address such questions. It does, however, affirm the utility of Dembski and Marks's ideas in testing the efficiency of a search function—an extremely important question in the context of evaluating Darwinian evolution.

- **Winston Ewert, W. A. Dembski and Robert J. Marks II, "Algorithmic Specified Complexity in the Game of Life," *Systems, Man, and Cybernetics: Systems, IEEE Transactions*, Vol. 45(4): 584-594 (April, 2015).**

This paper develops algorithmic specified complexity (ASC) as an improved method of measuring the functional meaning of biological (and other forms of) information and detecting design. The authors begin by observing that "Neither fundamental Shannon

nor Kolmogorov information models are equipped” to measure “meaningful” information. Complex and specified information (CSI) has long been cited as a method of measuring the functional meaning of information. ASC is a new flavor of CSI which can measure the degree to which information is meaningful:

The arranging of a large collection of parts into a working machine is highly improbable. However, *any* arrangement would be improbable regardless of whether the configuration had any functionality whatsoever. For this reason, neither Shannon nor KCS [Kolmogorov-Chaitin-Solomonoff] information models are capable of directly measuring meaning. Functional machines are specified—they follow some independent pattern. When something is both improbable and specified, we say that it exhibits *specified complexity*. An elaborate functional machine exemplifies high specified complexity. We propose a model, algorithmic specified complexity (ASC), whereby specified complexity can be measured in bits.

ASC is similar to KCS in that it assumes a computer environment which can describe some scenario in terms of commands in a programming language. This can allow, as they put it, a “quantitative measurement of specified complexity.”

To illustrate, they study patterns in the “Game of Life” computer simulation, such as “gliders” which move across the screen, or “Gemini,” a complex pattern which can copy itself. Ewert, Dembski, and Marks use these features of “Game of Life” to test the utility of ASC for detecting design. They find that some patterns are “simple enough that they arise from random configurations of cell space,” but “[o]thers required careful construction.” Their model predicts that high ASC patterns would arise by design, and that patterns which are known to appear randomly would have low ASC. They found that ASC is generally a good predictor of whether patterns appear at random or require design:

We have merely calculated the probability of generating the pattern through some simply random process not through the actual Game of Life process. We hypothesized that it was close enough to differentiate randomly achievable patterns from one that were deliberately created. This appears to work, with the exception of the unix pattern. However, even that pattern was less than an order of magnitude more probable than the bound suggested. This suggests the approximation was reasonable, but there is room for improvement.

We conclude that many of the machines built in the Game of Life do exhibit significant ASC. ASC was able to largely distinguish constructed patterns from those which were produced by random configurations. They do not appear to have been generated by a stochastic process approximated by the probability model we presented.

In other words, many high ASC patterns in Game of Life don't arise randomly. But is that surprising? After all, the "Game of Life" is a computer program created by intelligent agents that's designed to mimic living systems—systems which also have high ASC, and don't arise randomly. As they conclude, "Our work here demonstrates the applicability of ASC to the measure of functional meaning."

- **John Sanford, Wesley Brewer, Franzine Smith, and John Baumgardner, "The waiting time problem in a model hominin population," *Theoretical Biology and Medical Modelling*, Vol. 12:18 (2015).**

This paper cites the research of ID theorists Michael Behe, Douglas Axe, and Ann Gauger and uses a computer simulation of Darwinian evolution to address the question, "How long does it take for the simplest biological text strings to arise and be fixed, within a hominin population?" The authors begin by noting that "Given the unique capabilities of humans, an evolving hominin population would need to establish a great deal of new information, leading to new functionalities." They observe that Haldane's Dilemma suggests that very long time periods are required to fix multiple mutations that are required for some trait. Thus, "waiting for just the right mutation to arise in just the right location can be a rate-limiting factor in terms of the long-term evolution of any relatively small population" and "the generation and fixation of multiple specific mutations needed to combine to create a new function can require inordinately long waiting times." After favorably reviewing the work of Behe, Axe, Gauger, and others, the paper seeks to take a "fresh approach" and "use biologically realistic numerical simulations to analyze waiting times for the generation and fixation of specific strings of nucleotides of various lengths, given different mutation rates, given different selection pressures, and given different population sizes." The numerical simulation, Mendel's Accountant, allows a user to model an evolving population while modulating relevant different parameters of that population. After running the simulation, they find that "the waiting time problem is a significant constraint on the macroevolution of the classic hominin population" since "[r]outine establishment of specific beneficial strings of two or more nucleotides becomes very problematic." Indeed, they found that "For nucleotide strings of moderate length (eight or above), waiting times will typically exceed the estimated age of the universe – even when using highly favorable settings." They conclude: "To the extent that waiting time is a serious problem for classic neo-Darwinian theory, it is only reasonable that we begin to examine alternative models regarding how biological information arises."

- **Laurence A Cole, "The Evolution of the Primate, Hominid and Human Brain," *Journal of Primatology*, Vol. 4(1), DOI:10.4172/2167-6801.1000124 (2015).**

In this peer-reviewed paper, biochemist Laurence Cole argues that guided evolutionary processes were involved in the origin of the human brain. Cole who earned his PhD in biochemistry from the Medical College of Wisconsin and is a former faculty at Yale and the University of New Mexico, argues that larger brain sizes in humans and other primates were permitted by the development of a molecule called hyperglycosylated chorionic gonadotropin (HCG). In his view, the evolution of this molecule allowed

increased nutrients to pass through the placenta during human development, allowing primate brains to grow larger.

Cole offers a hypothesis of guided evolution of human brain enlargement where an intelligent agent—whom he identifies as “God”—was behind the process. His model requires that four specific evolutionary steps caused new biomolecules to arise which allowed increased nutrients passing through the placenta, which in turn allowed larger brain growth, which in turn allowed higher intelligence. Many other genes would have been involved in this process as well, and Cole appreciates that the whole evolutionary pathway—including these four specific events—are highly unlikely to occur by chance. He thus writes:

It is the evolution of CG and hyperglycosylated CG alone that led to the four clear steps in the development of the human brain and presented in the “CG/hyperglycosylated CG human evolution model”. Yes it is important that these primates' brains were continuously promoted by seven brain growth genes and their coded proteins. But brain growth, however, was only permitted by the evolution of forever improving promoters of hemochorial placentation and implantation, CG and hyperglycosylated CG.

Considering Darwin’s evolution model, the human evolution model described here is somewhat strange. Normally, positive mutations, such as those which occurred with CG and hyperglycosylated CG might take on 100 or more functions. Positive mutation may cause a hardening of a beak or mouth leading to better eating, a strengthening of any one of 50 muscles leading to increased strength, and improvement in liver enzyme functions, an improvement in vocal functions, better wiggling of toes, and so on. Furthermore, most mutation do not lead to positive outcomes. Mutation may not happen at all. As such the odds of a mutation in the CG gene leading to increased CG biological activity may be very small, perhaps 1 in 1000 or 1 in 10,000 offspring. In the “CG/hyperglycosylated CG human evolution model”, it appears like four mutation in the CG gene leading to major improvement in brain size occurred in a row, prosimian primate -(1)- lower simian primate -(2)- advanced simian primate -(3)- early hominids -(4)- humanoids. Four 1 in 1000 or 1 in 10,000 events occurring in a row appears like planned evolution rather than Darwinian evolution with remote odds of anywhere between 1 in a trillion and 1 in 10 quadrillion. This indicates that human brain development may have been planned rather than randomly evolved through Darwinian evolution. In this respect “the CG/hyperglycosylated CG human evolution model” could be suggestive of God's involvement in planning human creation as indicated in the Bible.

This model of intelligently guided evolution reflects an understanding that the likelihood of many specific mutations occurring is too low to be feasible under Darwinian

evolution. The appearance of such models in the literature indicates that scientists are increasingly taking seriously the concept of intelligent design.

- **Maricclair A. Reeves, Ann K. Gauger, and Douglas D. Axe, “Enzyme Families—Shared Evolutionary History or Shared Design? A Study of the GABA-Aminotransferase Family,” *BIO-Complexity*, Vol. 2014 (4).**

When Michael Behe published *Darwin’s Black Box* in 1996, he outlined irreducible complexity as a biochemical challenge to Darwinian evolution. Evolutionists responded by claiming that irreducibly complex features can be built through co-option, where a gene may be duplicated, and then the extra copy borrowed and retooled, or “co-opted,” to perform some new function. This peer-reviewed research paper from protein scientists at Biologic Institute experimentally tests the co-option model, showing it's very difficult for proteins to evolve new functions.

The project began in 2011, when Biologic researchers Ann Gauger and Douglas Axe published results of laboratory experiments trying to convert one enzyme (Kbl₂) to perform the function of another enzyme (BioF₂). Because these two proteins have a similar structure and are members of the same family, they are thought to be very closely related. Converting one protein to perform the function of a closely related protein is the sort of change which ought to be easily accomplished under the co-option model. However, after trying multiple combinations of different mutations, Gauger and Axe found, “successful functional conversion would in this case require seven or more” mutations. This posed a severe problem for Darwinian evolution, since a 2010 paper by Axe found that features which would require more than two harmful mutations, or more than six neutral mutations, before providing an advantage could not arise in the entire history of the earth. Axe and Gauger's 2011 study only investigated the evolvability of two proteins. Now in this 2014 paper, Axe, Gauger, and biochemist Maricclair Reeves, present new research on additional proteins from the same family, showing that they too are not amenable to an evolutionary conversion.

Their experiments examined nine other closely related enzymes to see if it is possible to convert them to perform the function of BioF₂. They induced all possible single mutations in the nine enzymes, and many other combinations of mutations, to determine if the enzymes could “evolve” the BioF₂ function. They found that this co-option scenario would require at least four mutations to convert an enzyme to function like BioF₂, including mutations to duplicate the gene and over-express it. Given that some of these mutations (such as duplication) would initially pose a disadvantage, it would take some 10¹⁵ years for the necessary mutations to arise to co-opt a protein to function like BioF₂—over 100,000 times longer than the age of the earth. Clearly this is not a feasible evolutionary scenario, as they conclude: “when all laboratory experience with enzyme conversion is considered collectively in this light, it seems quite clear both that the classical recruitment explanation of enzyme diversity is severely undermined and that there is no credible evolutionary alternative.”

- **David Snoke, “Systems Biology as a Research Program for Intelligent Design,” *BIO-Complexity*, Vol. 2014 (3).**

This article reviews the field of systems biology and argues that it is far more compatible with intelligent design than with unguided evolution: “Opponents of the intelligent design (ID) approach to biology have sometimes argued that the ID perspective discourages scientific investigation. To the contrary, it can be argued that the most productive new paradigm in systems biology is actually much more compatible with a belief in the intelligent design of life than with a belief in neo-Darwinian evolution. This new paradigm in system biology, which has arisen in the past ten years or so, analyzes living systems in terms of systems engineering concepts such as design, information processing, optimization, and other explicitly teleological concepts. This new paradigm offers a successful, quantitative, predictive theory for biology. Although the main practitioners of the field attribute the presence of such things to the outworking of natural selection, they cannot avoid using design language and design concepts in their research, and a straightforward look at the field indicates it is really a design approach altogether.” After observing, “It has become an extremely productive paradigm in biology to look for biological systems that exhibit the properties of sophisticated engineered systems, i.e., ones that resemble methods developed by human engineers over the past few hundred years to accomplish complicated tasks,” Snoke lists various features in biology that have been found to function like goal-directed, top-down engineered systems. After recounting such seemingly engineered aspects of biology, of the kind that systems biology studies, Snoke asks why systems biology has done such a good job of identifying these features of biology. He finds that the success of systems biology can be attributed to the assumptions it makes. And what are those? Snoke provides a list of assumptions that overlaps neatly with many of the assumptions of intelligent design. For example, he argues that systems biology assumes “teleology,” which is to say “top-down” rather than “bottom up” design. As he puts it, systems biology assumes that biological systems were built “starting with a goal, and then working backwards to see what is needed and used to accomplish that goal.” Snoke even quotes from proponents of systems biology urging biologists to recognize “the much-neglected teleological side of molecular biology.” Snoke’s closing words neatly deflect the objections of critics: “Many have demanded that the intelligent design paradigm must come up with a successful, predictive, quantitative program for biology, but it seems that such a program already exists right under our noses.”

- **Jonathan Wells, “Membrane Patterns Carry Ontogenetic Information That Is Specified Independently of DNA,” *BIO-Complexity*, Vol. 2014 (2).**

With over 400 citations to the technical literature, this peer-reviewed article by Jonathan Wells demonstrates compellingly that embryogenesis depends on crucial sources of information that exist outside of the DNA. This ontogenetic information guides the development of an organism, but because it is derived from sources outside of the DNA, it cannot be produced by mutations in DNA. Jonathan Wells concludes that because the neo-Darwinian model of evolution requires that variation is produced by

DNA mutations, neo-Darwinism cannot account for the origin of such epigenetic and ontogenetic information that exists outside of DNA.

Wells begins by observing that for decades, biologists accepted the “central dogma” of molecular biology — without qualification — which claims genes encoded by DNA entirely determine an organism. This view essentially says “DNA makes RNA makes protein makes us.” Wells writes:

The emphasis on genetic programs owes much to evolutionary theory — specifically, to the modern synthesis of Darwinian evolution and Mendelian genetics. According to the modern synthesis, new heritable variations originate in genetic mutations. In a 1970 interview, Monod said that with the establishment of the central dogma, “and the understanding of the random physical basis of mutation that molecular biology has also provided, the mechanism of Darwinism is at last securely founded”.

No one doubts that DNA encodes RNA, and RNA is translated to make proteins, but many other sources of information can enter the process along the way that do not stem directly from information encoded in DNA. For example, Wells observes that some of the basic axes of organismal development are in place *before* the initiation of developmental gene regulatory networks (dGRNs): “Spatial anisotropies precede — and are causally upstream of — the embryo’s dGRNs.”

Another non-DNA form of information Wells identifies is the “sugar code,” determined by complex patterns of sugar molecules, called glycans, on membrane surfaces. These molecules can carry high amounts of information since “carbohydrates can form branching chains that are far more elaborate than linear chains of nucleotides and amino acids.”

Wells also explains that “ion currents, transmembrane voltage potentials and [Endogenous Electric Fields] play significant roles in ontogeny comes from artificially disrupting them in vivo and then observing the effects of their disruption on morphogenesis,” but the information determining these electric fields is not in the DNA.

Population genetics — the mathematical basis for modern neo-Darwinian theory — is predicated upon the view that traits are encoded in DNA, and mutations in DNA produce new traits for natural selection to act upon. But since many traits aren’t determined by DNA, mutations in DNA cannot produce those traits. The very basis of the theory falls apart. Wells explains:

As we have seen, however, the idea that embryo development is controlled by a genetic program is inconsistent with the biological evidence. Embryo development requires far more ontogenetic information than is carried by DNA sequences. Thus Neo-Darwinism is false.

This is cutting-edge biology — and Wells grounds it in hundreds of citations to the peer-reviewed literature. Papers like this show that when freed from the “central dogmas” of neo-Darwinian evolution, a theory of intelligent design can open up promising and fruitful avenues of research and thinking in biology.

- **Winston Ewert, “Complexity in Computer Simulations,” *BIO-Complexity*, Vol. 2014 (1).** Computer scientist Winston Ewert reviews the literature claiming to evolve irreducible complexity through evolutionary computer simulations and finds that “Behe’s concept of irreducible complexity has not been falsified by computer models.” After reviewing the models, including Avida, Ev, Steiner trees, geometric model, digital ears, and Tierra, Ewert finds that in many cases, the “parts” that compose the irreducibly complex systems are “too simple,” in that the programs are designed such that systems which the programs deem “functional” are very likely to evolve. “Almost all of the cases of proposed irreducible complexity consist of parts simple enough that a system of several components could be produced by chance, acting without selection. As such, they fail to demonstrate that their models can evolve irreducibly complex systems, especially on the scale of biological complexity,” he writes. This leads to a conundrum for evolutionary theorists. Since “Darwinian evolution is an ateleological process,” this means that “If a model is designed to assist the evolution of an irreducibly complex system, it is not a model of Darwinian evolution” and “Any decision in the construction of a model made with an eye towards enabling the evolution of irreducible complexity invalidates the model.” Ewert finds that this is precisely where many of these models fail. In the one case that a truly irreducibly complex system was found in a program, he found it was “designed as part of the ancestor used to seed the ... simulation,” and thus did not actually evolve. According to Ewert’s analysis, computational attempts to explain the evolution of irreducible complexity have “failed on a number of fronts”:

Two of the models fail to satisfy the knockout test, in that they maintain functionality after parts have been removed. Almost all of the models use parts that are trivially complex, on the order of an amino acid rather than a protein in complexity. None of the models attempt to show why the mechanism used necessarily requires its parts. Finally, some of the models have been carefully designed to evolve. Thus, none of the models presented have demonstrated the ability to evolve an irreducibly complex system.

He concludes, “The prediction of irreducible complexity in computer simulations is that such systems will not generally evolve apart from intelligent aid” and finds that this prediction “has thus far stood the test in computer models.”

- **Steinar Thorvaldsen and Peter Øhrstrøm, “Darwin’s Perplexing Paradox intelligent design in nature,” *Perspectives in Biology and Medicine*, Vol. 56 (1): 78-98 (Winter, 2013).**

This paper in a prominent medical journal asserts that Darwin himself did not completely reject intelligent design and encourages modern Darwinians to consider following the same path. According to the authors, though Darwin rejected Paley’s arguments for design, “he was never able to ignore the powerful experience of the beauty and complexity of an intelligently designed universe, as a whole.” Contrary to ID-critics who claim ID is a recent mutation of creationism, these authors observe:

The term “intelligent design” is not new. It was used and discussed by Charles Darwin (1809–1882) in the years immediately after the publication of his *On the Origin of Species by Means of Natural Selection* (1859). He applied the term in an 1861 letter to Sir John F.W. Herschel (1792–1871).

But they were not the first to use the term as “Darwin and Herschel are likely to have got the term ‘intelligent design’ from Professor William Whewell of Trinity College, Cambridge (1794–1866), who seems to have been the first to use it.” They quote Whewell writing in 1833 nature shows “clear evidence of intelligent design, of arrangement with a benevolent end.” Indeed they observe that “the conversations regarding ‘design in nature’ are much older, dating back to the Greeks.”

The authors seek to propose ways to blend Darwin’s idea with intelligent design, noting that “neither Darwin nor any of his contemporaries found it unscientific when [Asa] Gray stated that ‘variation has been led along certain beneficial lines,’” where Gray “accepted natural selection as the cause of new species, but he did not believe it to be the only cause of variation, which he considered to be initiated by some inherent power, imparted in the beginning by divine design.” In their view, Darwin himself did not reject all forms of teleology in nature, arguing:

Darwin made a distinction between two kinds of intelligent design, one general (or cosmological), and one specific (related to the individual species). He accepted the former as a basis for a reasonable understanding of the origin of the universe, whereas he rejected the latter as relevant for a proper understanding of the living world. ... For Darwin himself, the idea of a divine designer was not the problem. In fact, he had nothing against the view that universe as a whole was intelligently designed, a notion that was part of the common worldview. However, the idea of a detailed, intelligent design was in conflict with his theory of natural selection.

They argue “both sides of the modern debate can benefit significantly by investigating the arguments and views formulated in the intelligent design debate of the 1860s and 1870s.” Specifically, they propose:

[B]oth sides may find it clarifying to refer to Darwin’s distinction between the two kinds of intelligent design. The critics of intelligent design should take into consideration that the other side actually has an interesting argument when they appeal to teleology, conceived as “teleology with teleology” (Brenner 2012), and modern advocates of intelligent design should understand why their view is considered provocative.

While ultimately the authors do not directly take a position on design in nature, they argue that the debate over design is a legitimate one which, in their view, “has yet to be generally settled.”

- **Winston Ewert, William A. Dembski, Robert J. Marks II, “Active Information in Metabiology,” *BIO-Complexity*, Vol. 2013 (4).**

The authors analyze “metabiology,” a field developed by the Argentine-American computer scientist and mathematician Gregory Chaitin, to use mathematics and computer simulations to formally prove that Darwinian mechanisms can create new information. Metabiology uses a gene-centered model of evolution, where a simulated “genetic code” (a hypothetical computer program) can be “mutated,” and when the program “halts” or stops running, it outputs a number that correlates with the program's fitness. If the number goes up as the evolutionary simulation proceeds, this is said to show Darwinian processes can create new information. Chaitin calls metabiology an “answer” to David Berlinski’s “stinging critique of Darwinism,” but the paper’s authors find the program deviates from biological reality, requiring informational inputs donated by an intelligent source—called “active information”—and does not truly demonstrate that unguided processes can produce new information.

Significantly, the paper finds that metabiology “pays no attention to resource limitations” and grants itself “unbounded resources and unbounded time,” thereby failing to adequately model real-world biological processes where probabilistic resources (e.g., time and population sizes) are limited. As they put it, “Metabiology’s math obscures the huge amounts of time required for the evolutionary process.” A related, unrealistic aspect of metabiology that it can systematically simulate all possible programs, which in effect allows it to completely rewrite its evolving program instantly. Such a process would never happen to genomes in biological organisms, meaning metabiology unrealistically grants access to the equivalent of unlimited computing resources.

The authors also explain that metabiology uses a halting oracle as a source of “active information.” A halting oracle is a hypothetical meta-program that can indicate whether a given program will ever stop running. They note Chaitin admits that the halting oracle in metabiology, “is where all the creativity is really coming from in our program,” but also he admits that such an oracle is “mathematical fantasy.” The authors thus aptly observe: “A computer tool proven not to exist is admittedly at the outset an obvious major strike against a theory purporting to demonstrate reality.” They conclude:

In order for evolution to occur in these models, external knowledge must be imposed on the process to guide it. Metabiology thus appears to be another example where its designer makes an evolutionary model work. ... Consistent with the laws of conservation of information, natural selection can only work using the guidance of active information, which can be provided only by a designer.

Properly understood, these programs show that evolution requires intelligent design.

- **Michael J. Denton, “The Types: A Persistent Structuralist Challenge to Darwinian Pan-Selectionism,” *BIO-Complexity*, Vol. 2013 (3).**

In this paper, Michael Denton challenges the view that biological organisms are accidents of random mutation and natural selection but instead adopts a structuralist viewpoint, where body plans are like Platonic “types,” programmed into the fabric of nature. According to Denton, this view, popular before Darwin wrote *Origin of Species* in 1859, “was supported by two fundamental observations: that the homologies appeared to be *non-adaptive abstract patterns*, and that in some cases they appear to have remained *invariant for hundreds of millions of years* in diverse lineages.” As examples of persistent, non-adaptive patterns, he cites the pentadactyl limb structure in vertebrates, the insect body plan, or the pentamerous symmetry of echinoderms. Denton contends that structuralism can account for these non-adaptive features that pervade life.

The Darwinian view, in contrast, is “functionalist,” wherein “organisms are in essence like machines, complexes of functional parts arranged to serve particular adaptive ends.” A structuralist view does not deny that adaptations exist, as structuralism “implies that organic order is a mix of two completely different types of order, generated by two different causal mechanisms: a primal order generated by natural law, and a secondary adaptive order imposed by environmental constraints (by natural selection according to Darwinists, by Lamarckian mechanisms and by intelligent design according to current design theorists).” But Denton argues that the functionalism required by Darwinian evolution cannot account for seemingly non-adaptive features. This is a major problem for Darwinism, as Denton cites “a vast universe of non-adaptive forms and patterns in nature which no biologist, not even the most convinced functionalist or Darwinist, has ever claimed to serve specific adaptive functions.” Thus, “neither Darwin nor any subsequent Darwinist has ever provided cogent reasons for accepting the grand claim that all complexity in biology (including all currently non-adaptive forms) has resulted from past adaptive and purposeful shaping of structures to serve functional ends.”

So what can explain the origin of these features? Denton proposes that a structuralist view can be rehabilitated since “during the 20th century several advances in different fields have provided new support for the pre-Darwinian idea of life and its deep structures as immanent in the world order.” These discoveries include the fine-tuning of

the universe for life, covered by Denton in a previous paper (*BIO-Complexity*, Vol. 2013 (1).) Here, Denton adds some new parameters from biochemistry:

- *DNA*: The chemical stability of the double-helical shape of DNA which allows it to “perform one of the most important of biological functions,” including the fact that “its base sequence may contain ‘complex specified information’.”
- *Protein folding*: “the rules that generate the thousand-plus known protein folds have now been largely elucidated and remarkably they amount to a set of ‘laws of form’ of precisely the kind sought after by early 19th-century biologists.”
- *Lipids*: Lipid membranes “arise mainly from the self-organization of the membranes themselves, by energy minimization without any direction from anything like a genetic blueprint.”
- *Microtubules*: According to Denton, “The microtubular aster is another example of a molecular form that clearly arises directly out of the intrinsic self-organizing properties of its basic constituents.”

Denton explains that the specification of these structures in the genetic code is not enough to explain their functionality, and “Rather, in every case the primary natural self-organizing propensity of a particular category of matter is exploited and secondarily modified to serve some adaptive end.” He likewise suggests that cell form, and even organismal form, might heavily depend on natural laws to take their shapes. In his view, “organic form at all levels of the biological hierarchy, not just at the cellular level, is essentially emergent and epigenetic, arising from complex dynamic self-organizing mechanisms during development.”

Denton concludes that: “After 150 years of focused functionalist effort, the grand taxonomic system and the ascending hierarchy of homologous patterns has still not been adequately accounted for in functionalist/adaptive/Darwinian terms.” The structuralist view of biology he proposes is not exactly the same as intelligent design, but it’s quite compatible with a designed universe, where the laws of nature are finely-tuned to allow for complex life to exist. Just as Darwinism cannot explain these laws, these laws cannot explain all the adaptive complexity of life. Structuralist views leave plenty of room for intelligent design.

- **Stephen C. Meyer, *Darwin's Doubt: The Explosive Origin of Animal Life and the Case for Intelligent Design* (HarperOne, 2013).**

Charles Darwin knew that there was a significant event in the history of life that his theory did not explain. In what today is known as the “Cambrian explosion,” 530 million years ago, many animals suddenly appeared in the fossil record without apparent ancestors in earlier layers of rock. In *Darwin’s Doubt*, Stephen C. Meyer tells the story of the mystery surrounding this explosion of animal life—a mystery that has intensified, not only because the expected ancestors of these animals have been not found, but also because scientists have learned what it takes to construct an animal. Meyer argues that the theory of intelligent design—which holds that certain features of the universe are

best explained by an intelligent cause, not an undirected cause such as natural selection—is ultimately the best explanation for the origin of the Cambrian explosion. The publisher, HarperOne, conducted an external peer-review with two distinguished paleontologists and two evolutionary biologists.

- **Granville Sewell, “Entropy and Evolution,” *BIO-Complexity*, Vol. 2013 (2).**

In this paper, mathematician Granville Sewell explores whether the second law of thermodynamics poses difficulties for Darwinian evolution. Some early critics of Darwinism argued that the second law “must” pose a problem because evolutionary models require a decrease in entropy. Sewell avoids such unsophisticated arguments, but nonetheless reframes the issue to show that the second law *could potentially* be an obstacle.

Sewell observes that evolutionists responded to older second-law criticisms via a “compensation argument,” claiming that “because the Earth is an open system” that therefore “entropy can decrease in an open system, provided the decrease is compensated by entropy increases outside the system.” Sewell is skeptical of that rejoinder and points out that:

the fact that order can increase in an open system does not mean that tornados can turn rubble into houses and cars without violating the second law. And it does not mean that computers can appear on a barren planet as long as the planet receives solar energy. Something must be entering from outside which makes the appearance of computers not extremely improbable, for example, computers.

Can unguided natural causes provide the “something” to produce the kind of order required for life. Sewell again explains why the “compensation argument” fails:

In an open system you just have to take into account what is entering (and leaving) the system when deciding what is extremely improbable and what is not. When thermal entropy decreases in an open system, there is not anything macroscopically describable happening that is extremely improbable from the microscopic point of view; rather, something is crossing the boundary that makes the decrease not extremely improbable.

He would thus formulate the following rule: “Natural (unintelligent) forces do not do macroscopically describable things that are extremely improbable from the microscopic point of view.” What this means is that given the second law of thermodynamics, the compensation argument does not necessarily solve the problem for Darwinian evolution, and the second law *could* potentially be a problem for Darwinism. If Darwinian advocates were willing to candidly examine the improbabilities faced by their theory, they would see that serious questions about the second law -- among many others, of course -- remain to be answered.

- **Michael J. Denton, “The Place of Life and Man in Nature: Defending the Anthropocentric Thesis,” *BIO-Complexity*, Vol. 2013 (1).**

In this paper, Michael Denton argues that the “the order of nature is uniquely suitable for life as it exists on earth (Terran life), and specifically for living beings similar to modern humans.” He opens by observing that after “the Copernican revolution and particularly after the publication of Darwin’s *On the Origin of Species* ... mankind, so it seemed, had no special place in nature.” But this article reviews “discoveries in chemistry and biochemistry” of the past 100 years which “have reopened the ‘grand debate’ by providing intriguing new support for the old and seemingly obsolete anthropocentric paradigm.” However, “To make the radical claim that the universe is designed for our existence,” Denton observes, we must demonstrate “a cosmos where the laws of nature are uniquely fit for Terran life” rather than “exotic biochemistries” like “Star Treklike aliens.” Toward this end, Denton observes that many of the basic chemical constituents on Earth -- water, carbon dioxide, oxygen and organic compounds – are specially fit for life as we know it:

- **Carbon:** According to Denton, “carbon is unique in its ability to combine with other atoms, forming a vast and unparalleled number of compounds in combination with hydrogen, oxygen and nitrogen.” Moreover “the general ‘metastability’ of carbon bonds and the consequent relative ease with which they can be assembled and rearranged by living systems contributes greatly to the fitness of carbon chemistry for biochemical life,” and means “that no other atom is nearly as fit as carbon for the formation of complex biochemistry.”
- **Water:** Denton observes that water is “is able to hold in solution an enormous, unequalled range of diverse chemical compounds” and thus has a power as a “far greater than that of almost any other common fluid.” It has also been discovered that “the temperature range in which water is a fluid, 0–100°C, overlaps with the temperature range in which chemical bonds can be readily manipulated by biochemical system.” These properties form an essential role in protein folding and the formation of the cell membrane.
- **Carbon dioxide:** CO₂ is special because it “not only distributes carbon to all corners of the hydrosphere, it also maintains the acid-base balance of the hydrosphere, generating a controlled aqueous environment in which the carbon it distributes can be assembled into living systems.” In Denton’s view, “No less than water, then, CO₂ is uniquely fit for carbon-based life.”
- **Oxygen:** Denton points out that “The fact that oxidations, particularly of carbon and hydrogen, provide more energy than nearly all other types of chemical reactions is of particular importance.”

Denton also cites “‘cosmic coincidences,’ the notion that the fundamental physical constants that determine the overall structure of the universe and the laws of nature must be very close to the values observed to generate a universe capable of harboring life.” For example, “the ‘lucky’ fact that the nuclear resonances of the isotopes ¹²C and ¹⁶O are exactly what they need to be if carbon is to be synthesized and accumulate in

any quantity in the interior of stars.” He further finds that these finely-tuned parameters are more important for complex human physiology than simpler life, such as bacteria.

Denton closes by asking, “Can we infer that anthropocentric fine-tuning is the result of intelligent design?” He argues that “it is very hard not to be struck by the fact that the properties of the members of the vital ensemble are peculiarly fit for life as it is on earth, in a profoundly synergistic and parsimonious way,” and thus “new discoveries in organic chemistry and biochemistry, unrecognized at the time, were providing the first hint that life on earth might after all be the result of design.”

- **Berkley E. Gryder, Chase W. Nelson, and Samuel S. Shepard, “Biosemiotic Entropy of the Genome: Mutations and Epigenetic Imbalances Resulting in Cancer,” *Entropy*, 15: 234-261 (2013).**

ID encourages scientists to understand biological systems like designed objects. This paper thus shows how ID proponents can apply ID thinking to help approach scientific problems, like the causes of cancer. Using this approach, this paper compares living organisms to semiotic – i.e., symbol or language-based – systems in order to understand cancer. It observes:

Recognizing living organisms as semiotic systems allows for useful analogies to be drawn from other semiotic systems. Such analogies are powerful because: (1) they give insight and understanding by relating the unfamiliar in terms of the familiar and, (2) lessons learned from other semiotic spheres (such as principles of efficient information storage and retrieval in computer science) can generate predictions and hypotheses for new frontiers in biology (such as a tree-like database structure for information storage and retrieval in the human genome). This is evidenced by the fact that biologists frequently use analogies from the familiar semiotic systems of human language and computers.

They further observe: “The DNA message is read, copied, edited, transcribed, and translated. It is striking that the most fitting terms used to describe the biochemical mechanics of life are rooted not in biology, chemistry or mechanics, but rather, in language.” If the best analogies for biological systems are to designed objects, what does that say about the nature and origin of biological systems? How can this analogy help us deal with cancer? Since “cancer is a disease of genome alterations,” then it is a disease caused by “deterioration of biological sign systems,” which they call “biosemiotic entropy.” It is the breakdown in transmission of DNA’s language-based information – or biosemiotic entropy – which causes cancer. They conclude, “Understanding the existing biosemiotic systems within individuals, the parameters affecting their entropy, and their eventual deterioration leading to cancer may aid hypothesis generation for more effective treatments.”

- **Vladimir I. shCherbak and Maxim A. Makukov, “The ‘Wow! Signal’ of the terrestrial genetic code,” *Icarus*, Vol. 224 (1): 228-242 (May, 2013).**

This peer-reviewed paper in the respected scientific journal *Icarus* proposes that “patterns of symbolic language” in our DNA might contain an “intelligent signature.” Since “the actual scenario for the origin of terrestrial life is far from being settled,” the paper argues, “the proposal that it might have been seeded intentionally cannot be ruled out.” Their proposed methods for detecting design are not entirely dissimilar from those commonly proposed by ID proponents:

To be considered unambiguously as an intelligent signal, any patterns in the code must satisfy the following two criteria: (1) they must be highly significant statistically and (2) not only must they possess intelligent-like features, but they should be inconsistent in principle with any natural process, be it Darwinian or Lamarckian evolution, driven by amino acid biosynthesis, genomic changes, affinities between (anti)codons and amino acids, selection for the increased diversity of proteins energetics of codon-anticodon interactions, or various pre-translational mechanisms.

The authors seem to argue for a natural, extraterrestrial intelligence, as they posture their argument as similar to a biological version of SETI. Nonetheless, the fact remains that this paper is attempting to argue for design detection within biology, placing it within ID thinking proper.

- **Winston Ewert, William A. Dembski and Robert J. Marks II, “Conservation of Information in Relative Search Performance,” *Proceedings of the 2013 IEEE 45th Southeastern Symposium on Systems Theory (SSST)*, Baylor University, March 11, 2013, pp. 41-50.**

According to this paper, the “No Free Lunch” theorems studied by William Dembski predict that “all search algorithms have the same performance on the average.” Some might argue that when search performance is compared in a relative manner, “Some algorithms look to perform better than others.” The authors find, however, that this claim does not hold, once you examine the average performance across related searches:

[T]his advantage is lost when averaging is over a group of related algorithms. Every advantage against one algorithm is balanced by a disadvantage against a related algorithm.

The investigators consider the example of three treasure-hunters on an island, each searching in different places. One treasure-hunter might be able to look at the empty holes dug by another to learn that the treasure wasn’t found in that location, thereby improving his search performance. Some “searches” might have more information about the target than others, but when you take the average of the treasure-hunters

performance across the entire island, they collectively perform no better than a random search. The authors conclude:

In order to best an algorithm, active information is required from extra knowledge of the problem. This parallels the case of the NFL requiring information about the fitness function in order to improve performance. Finally, random search exhibits the same average performance regardless of which algorithm it faces. Thus no way exists to gain an advantage on average over random search. The principle of conservation of information still applies in the case of relative performance metrics. The appearance of free lunches in relative performance metrics does not give us any way to exploit them to create generally superior optimizers.

The implication is that Darwinian evolution, on average, can't ever perform better than a random search.

- **Winston Ewert, William A. Dembski and Robert J. Marks II, "On the Improbability of Algorithmically Specified Complexity," *Proceedings of the 2013 IEEE 45th Southeastern Symposium on Systems Theory (SSST)*, Baylor University, March 11, 2013, pp. 68-70.** The classical formulation of detecting design seeks to find complexity and specification. In other work, the Evolutionary Informatics Lab has developed algorithmic specified complexity (ASC) as a method of quantifying specification. But can ASC be a measure of the probability, or complexity of an event as well? In this paper, the authors "show a bound on the probability of obtaining a particular value of algorithmic specified complexity." They conclude that "high ASC objects are improbable," and useful for detecting design – objects which they call "special" compared to normal natural events.

ASC incorporates the concept of Kolmogorov complexity, defined as the "shortest computer program length required to reproduce a specified bitstring description of an object." This metric allows the authors to identify objects which are unlikely, but also follow predictable patterns:

The usefulness of this definition depends on the wide variety of constructs that are compressible. This includes for example simple pattern, such as "01" repeated 32 times. It also includes valid English text, which given a knowledge of the English language can be compressed. It also include complex functioning systems because they can be described by their functionality rather than the system that produces that functionality. Thus Kolmogorov complexity captures a wide variety of objects that we deem "special." Thus we can usefully apply this metric to a wide variety of objects.

They conclude, "that an object exhibiting high ASC is unlikely to arise," thus fulfilling part of the criteria for detecting design.

- **Winston Ewert, William A. Dembski, Ann K. Gauger, Robert J. Marks II, “Time and Information in Evolution,” *BIO-Complexity*, Vol. 2012 (4).**

This paper responds to a 2010 paper in *Proceedings of the U.S. National Academy of Sciences (PNAS)* titled “There’s plenty of time for evolution,” by Herbert S. Wilf and Warren J. Ewens, a biologist and a mathematician at the University of Pennsylvania. There’s little doubt that Wilf and Ewens intended to respond to ID arguments. Though strategically lacking any citations to ID literature, their *PNAS* paper’s abstract starts by stating, “Objections to Darwinian evolution are often based on the time required to carry out the necessary mutations,” and arguing that “there has been ample time for the evolution that we observe to have taken place.” Dembski, Gauger, and Marks then offer a long list of reasons why the Wilf and Ewens model of evolution isn’t biologically realistic because “within their model are implicit information sources, including the equivalent of a highly informed oracle that prophesies when a mutation is ‘correct,’ thus accelerating the search by the evolutionary process.” They also find that Wilf and Ewens “simplify the search” and incorrectly assume “no epistasis between beneficial mutations, no linkage between loci, and an unrealistic population size and base mutation rate, thus increasing the pool of beneficial mutations to be searched.” In effect, Wilf and Ewens ignore the problem of non-functional intermediates, wrongly assuming that all intermediate stages will be functional, or lead to some functional advantage. Because of these, and other problems, the authors argue, Wilf and Ewens’ “conclusion that there’s plenty of time for evolution is unwarranted.”

- **Matti Leisola, Ossi Pastinen, and Douglas D. Axe, “Lignin -- Designed Randomness,” *BIO-Complexity*, Vol. 2012 (3).**

We are used to thinking of design as a positive presence. What if the things we don't see are missing for a reason also? Lignin, a complex organic polymer found in wood, is the second most abundant biopolymer on the planet, and higher gram for gram in stored energy than the most abundant biopolymer, cellulose. Yet nothing living can directly use lignin as an energy source. Why? In this peer-reviewed paper, the authors argue that the answer is because the indigestibility of lignin may be an essential requirement for the balance of life. Lignin is an essential component of wood, but its indigestibility slows the degradation of wood, thus allowing the buildup of humus in the soil, which in turn permits plant growth and all resulting life that depends on plants. This paper thus extends design arguments into the realm of ecology.

The authors begin by recognizing that lignin poses a conundrum for Darwinism. Neo-Darwinian theory claims that new molecular functions readily evolve but this means:

The Darwinian account must somehow reconcile 400 million years of failure to evolve a relatively modest innovation—growth on lignin—with a long list of spectacular innovations thought to have evolved in a fraction of that time.

They thus ask: “How can microorganisms have failed to exploit lignin as an energy source while much less evolvable species have, on innumerable occasions, acquired solutions to problems that appear to be considerably harder?” In their view:

That tension vanishes completely when the design perspective is adopted. Terrestrial animal life is crucially dependent on terrestrial plant life, which is crucially dependent on soil, which is crucially dependent on the gradual photo- and biodegradation of lignin. Fungi accomplish the biodegradation, and the surprising fact that it costs them energy to do so keeps the process gradual. The peculiar properties of lignin therefore make perfect sense when seen as part of a coherent design for the entire ecosystem of our planet.

They conclude that lignin makes an argument for not just design in microbiology, but also in ecology: “Perhaps the oddest aspect of this is that Darwin’s theory is unable to make sense of a situation that otherwise makes perfect sense. If life is the product of intelligent design, it stands to reason that the whole design must be considered—not just the functions of molecules and cells and tissues and organs and organisms, but also the functions of entire ecosystems, all the way up to the global ecosystem.”

- **Kirk K. Durston, David K.Y. Chiu, Andrew K.C. Wong, and Gary C.L. Li, “Statistical discovery of site inter-dependencies in sub-molecular hierarchical protein structuring,” *EURASIP Journal on Bioinformatics and Systems Biology*, Vol. 2012 (8).** In prior papers, pro-intelligent design biophysicist Kirk Durston and others have developed the concept of “functional complexity,” a method of measuring biological information similar to CSI of ID theory. This peer-reviewed paper builds on that work by applying a method which compares similar sites across homologous proteins to determine which amino acids are necessary for protein function. This method allows identification of new sites within the 3D structure of proteins that are vital for the protein’s function. This paper shows the utility of ID’s information-based approach to studying biomolecules, as the paper explains: “The ability to discover key residue–residue contacts, branches, and larger structural sub-domains within a protein through the *k*-modes analysis of the multiple sequence alignment will be a significant asset in understanding the details in the sequence of protein folding, structure, and functionality among different residue locations within a hierarchical global protein framework. Furthermore, by discovering the important attribute clusters within a protein, predictions can also be made as to which mutations could be more harmful or more stable than others. All these play an important role in furthering our understanding of the information processing capability of genes and proteins, in terms of the specific use of functional units at specific locations on the sequence to create the 3D structure as well as the internal and external functionality of the molecules.”

- **Fernando Castro-Chavez, “A Tetrahedral Representation of the Genetic Code Emphasizing Aspects of Symmetry,” *BIO-Complexity*, Vol. 2012 (2).**

The beauty of the organization of the periodic table has long been recognized as an artifact of the aesthetic design embedded in nature. This article by Fernando Castro-Chavez, of the Department of Medicine at Baylor College of Medicine, develops new methods of visualizing the organization of the genetic code. Much like the periodic table finds there are geometric patterns to the properties of elements, Castro-Chavez proposes that “a geometric representation of the code will only be as compelling as the harmony between the chosen geometry and the biological reality.” He uses a tetrahedral shape to organize the code, where 16 of the 64 codons appear one each side. The four endpoints of the tetrahedron represent the start and stop codons, with the inner four triangles representing codons that encode hydrophobic amino acids, and wherever possible, “amino acids are grouped by salient properties.” He proposes this beautiful arrangement can lead to “new insights” into the code.

- **Winston Ewert, William A. Dembski, and Robert J. Marks II, “Climbing the Steiner Tree—Sources of Active Information in a Genetic Algorithm for Solving the Euclidean Steiner Tree Problem,” *BIO-Complexity*, Vol. 2012 (1).**

ID-critics like mathematician David Thomas have argued that genetic algorithms mimicking natural selection can solve the Steiner tree problem, a classic problem in evolutionary computation which seeks to find the shortest pathway interconnecting a set of points. In this paper, researchers at the Evolutionary Informatics Lab argue that intelligence is necessary to solve problems like the Steiner tree. The authors explain that, “A genetic algorithm is a search algorithm that uses procedures that mimic natural selection and random mutation to determine which candidate solutions to try next,” but their research has developed a method of determining how much “active information” has been “through incorporating sources of information derived from the programmer’s prior knowledge.”

They respond to Thomas arguing that “Thomas is under the misapprehension that intelligent design advocates claim that the actual answer is encoded into the algorithm,” whereas “This is not in fact what intelligent design advocates claim.” Rather, the claim is that programmers can import active information into programs in more subtle ways, using prior knowledge to fine-tune the algorithm to find the solution. While this might be a good programming strategy, it is nothing like the blind and unguided process of Darwinian evolution since “success is due to prior knowledge being exploited to produce active information in the search algorithm.” They conclude, “Only a teleological process guided by some form of intelligence can function in this way. Insofar as simulations of evolution make use of prior knowledge, they are not simulations of Darwinian evolution in any meaningful sense.”

- **Joseph A. Kuhn, “Dissecting Darwinism,” *Baylor University Medical Center Proceedings*, Vol. 25(1): 41-47 (2012).**

This article by Dr. Joseph Kuhn of the Department of Surgery at Baylor University Medical Center appeared in the peer-reviewed journal *Baylor University Medical Center Proceedings*. It poses a number of challenges to both chemical and biological evolution, including:

1. Limitations of the chemical origin of life data to explain the origin of DNA
2. Limitations of mutation and natural selection theories to address the irreducible complexity of the cell
3. Limitations of transitional species data to account for the multitude of changes involved in the transition.

Regarding the chemical origin of life, Kuhn points to the Miller-Urey experiments and correctly observes that “the experimental conditions of a low-oxygen, nitrogen-rich reducing environment have been refuted.” Citing Stephen Meyer’s *Signature in the Cell*, he contends that “the fundamental and insurmountable problem with Darwinian evolution lies in the remarkable complexity and inherent information contained within DNA.” Kuhn also explains that “Darwinian evolution and natural selection could not have been causes of the origin of life, because they require replication to operate, and there was no replication prior to the origin of life,” but no other known cause can organize the information in life.

Dr. Kuhn then turns to explaining the concept of irreducible complexity, citing Michael Behe’s book *Darwin’s Black Box* and noting that “irreducible complexity suggests that all elements of a system must be present simultaneously rather than evolve through a stepwise, sequential improvement, as theorized by Darwinian evolution.” Further, “The fact that these irreducibly complex systems are specifically coded through DNA adds another layer of complexity called ‘specified complexity.’” As a medical doctor, Kuhn proposes that irreducibly complex systems within the human body include “vision, balance, the respiratory system, the circulatory system, the immune system, the gastrointestinal system, the skin, the endocrine system, and taste.” He concludes that “the human body represents an irreducibly complex system on a cellular and an organ/system basis.”

Kuhn also explores the question of human/ape common ancestry, citing Jonathan Wells’s book *The Myth of Junk DNA* and arguing:

DNA homology between ape and man has been reported to be 96% when considering only the current protein-mapping sequences, which represent only 2% of the total genome. However, the actual similarity of the DNA is approximately 70% to 75% when considering the full genome, including the previously presumed “junk DNA,” which has now been demonstrated to code for supporting elements in

transcription or expression. The 25% difference represents almost 35 million single nucleotide changes and 5 million insertions or deletions.

In Dr. Kuhn's view, this poses a problem for Darwinian evolution because the "[t]he ape to human species change would require an incredibly rapid rate of mutation leading to formation of new DNA, thousands of new proteins, and untold cellular, neural, digestive, and immune-related changes in DNA, which would code for the thousands of new functioning proteins."

Kuhn also observes that a challenge to neo-Darwinism comes from the Cambrian explosion:

Thousands of specimens were available at the time of Darwin. Millions of specimens have been classified and studied in the past 50 years. It is remarkable to note that each of these shows a virtual explosion of nearly all phyla (35/40) of the animal kingdom over a relatively short period during the Cambrian era 525 to 530 million years ago. Since that time, there has been occasional species extinction, but only rare new phyla have been convincingly identified. The seminal paper from paleoanthropologists J. Valentine and D. H. Erwin notes that the absence of transitional species for any of the Cambrian phyla limits the neo-Darwinian explanation for evolution.

Despite Texas's call for discussing the scientific strengths and weaknesses of Darwinian evolution, Kuhn closes by noting, "In 2011, when new textbooks were presented to the State Board of Education, 9 out of 10 failed to provide the mandated supplementary curricula, which would include both positive and negative aspects of evolution (44)." Citing [Discovery Institute's Report on the Texas Textbooks](#), he laments:

[S]everal of the textbooks continued to incorrectly promote the debunked Miller-Urey origin of life experiment, the long-discredited claims about nonfunctional appendix and tonsils, and the fraudulent embryo drawings from Ernst Haeckel. In essence, current biology students, aspiring medical students, and future scientists are not being taught the whole story. Rather, evidence suggests that they continue to receive incorrect and incomplete material that exaggerates the effect of random mutation and natural selection to account for DNA, the cell, or the transition from species to species.

Kuhn concludes, "It is therefore time to sharpen the minds of students, biologists, and physicians for the possibility of a new paradigm."

- **David L. Abel, "Is Life Unique?," *Life*, Vol. 2:106-134 (2012).**
What is it that distinguishes life from non-living entities? This peer-reviewed paper attempts to answer that question, noting that "Life pursues thousands of biofunctional goals," whereas "Neither physicydynamics, nor evolution, pursue goals." Is it possible

that unguided evolution and strictly material causes produced life's purposeful processes? According to this paper, the answer is no. Life's goals include the use of "symbol systems" to maintain "homeostasis far from equilibrium in the harshest of environments, positive and negative feedback mechanisms, prevention and correction of its own errors, and organization of its components into Sustained Functional Systems." But the article notes that "the integration and regulation of biochemical pathways and cycles into homeostatic metabolism is programmatically controlled, not just physicomatically constrained." This programming is termed "cybernetic"—yet according to the paper cybernetic control "flows only from the nonphysical world of formalism into the physical world through the instantiation of purposeful choices." Indeed, "Only purposeful choice contingency at bona fide decision nodes can rescue from eventual deterioration the organization and function previously programmed into physicality." Life thus cannot be the result of unguided material processes—some cause capable of programming "purposeful choices" is necessary.

- **Douglas D. Axe, Philip Lu, and Stephanie Flatau, "A Stylus-Generated Artificial Genome with Analogy to Minimal Bacterial Genomes," *BIO-Complexity*, Vol. 2011 (3).**

This peer-reviewed paper is a follow-up up to the 2008 *PLoS One* paper co-authored by Axe and Lu on *Stylus*, a computer simulation of evolution which is more faithful to biological reality than many others. This 2011 paper explains that the "functions" of the digital organisms in other simulations are often divorced from real-world meaning. They designed *Stylus* to present a more accurate picture:

The motivation for *Stylus* was the recognition that prior models used to study evolutionary innovation did not adequately represent the complex causal connection between genotypes and phenotypes.

Stylus aims to correct these deficiencies by simulating Darwinian evolution in a manner that more accurately reflects the biological relationship between genotype and phenotype. It is also more realistic because it solves real-world problems. As the paper explains, "Functional specificity therefore has a structural basis in the *Stylus* world, just as it does in the real world." *Stylus* manipulates digital objects that have real-world meaning: the targets of evolution in *Stylus* are Chinese characters. As the paper explains:

These translation products, called vector proteins, are functionless unless they form legible Chinese characters, in which case they serve the real function of writing. This coupling of artificial genetic causation to the real world of language makes evolutionary experimentation possible in a context where innovation can have a richness of variety and a depth of causal complexity that at least hints at what is needed to explain the complexity of bacterial proteomes.

These characters not only have real-world meaning, but their function-related shapes bear interesting analogies to proteins. An additional similarity between Chinese

characters and proteins is that just as protein domains are re-used throughout many proteins, so particular shapes, called “strokes,” are found commonly throughout Chinese characters.

Basic to life is an information conversion, where the information carried in genes (the genotype) is converted into an organism’s observable traits (the phenotype). Those biological structures then perform various functions. Another way of framing this information conversion is therefore: sequence → structure → function. Axe, Lu and Flatau explain that many previous computer programs attempting to simulate evolution achieve part of this conversion, but not the whole thing.

For example, Conway’s famous Game of Life starts with a structure, and in some instances that structure can perform a function. But there is no sequence involved in the conversion. Avida starts with a sequence of programming commands, and when successful performs certain logic functions. But in Avida there is no structure to mediate between sequence and function. *Stylus*, on the other hand, is more advanced in that it simulates the full sequence → structure → function information transfer. It does this by starting with a programmed genome. As the paper explains:

[The] *Stylus* genome encodes a special kind of text, namely, one that describes how to decode the genome. That is, the desired genome will encode a sequence of Chinese characters (in the form of vector proteins) that tells a reader of Chinese how *Stylus* genes are translated into vector sequences, and how those sequences are processed to make readable vector proteins.

The paper explains: “What *Stylus* offers that no other model offers, to our knowledge, is an artificial version of gene-to-protein genetic causation that parallels the real thing.”

In the world of *Stylus*, a Chinese character is like a protein. So how can we determine if a functional “protein” has evolved? According to the paper, “At the core of *Stylus* software is an algorithm that quantifies the likeness of a given vector protein to a specified Chinese character.” This complicated algorithm is described as follows:

Stylus endows these graphical constructs with interesting similarities to their molecular counterparts by uncovering and exploiting a pre-existing analogy -- the analogy between the set of characters used in Chinese writing and the set of protein structures used in life. Specifically, vector proteins are drawn objects that may function as legible Chinese characters if they are suitably formed. ... *Stylus* is unique in its use of real function that maps well to molecular biology. It therefore represents a significant advance in the field of evolutionary modeling. (internal citations omitted)

The paper presents a set of Chinese characters that can be used for simulating the evolutionary process in the *Stylus* world. But can these Chinese character groups, which

have many qualities that parallel real-world protein families, evolve by random mutation and natural selection? That's the sort of question the creators of *Stylus* hope to answer. The results of such simulations will probably be fleshed out in future papers. But the current paper leaves us with a strong sense of where this is all heading:

Evolutionary causation is intrinsically tied to the relationship between genotype and phenotype, which depends on low-level genetic causation. It follows that evolutionary explanations of the origin of functional protein systems must subordinate themselves to our understanding of how those systems operate. In other words, the study of evolutionary causation cannot enjoy the disciplinary autonomy that studies of genetic causation can.

In view of this, the contribution of *Stylus* is to make evolutionary experimentation possible in a model world where low-level genetic causation has the essential role that it has in the real world. Combined with the free *Stylus* software, the complete *Stylus* genome made freely available with this paper paves the way for analogy-based studies on a wide variety of important subjects, many of which are difficult to study by direct experimentation. Among these are the evolution of new protein folds by combining existing parts, the optimality and evolutionary optimization of the genetic code, the significance of selective thresholds for the origin and optimization of protein functions, and the reliability of methods used for homology detection and phylogenetic-tree construction.

There probably will never be a perfect computer simulation of biological evolution, but *Stylus* brings new and improved methods to the field of evolutionary modeling. This tool will help those interested in testing the viability of Darwinian claims to assess whether complex features can be created by random mutations at the molecular level.

- **Stephen C. Meyer and Paul A. Nelson, “Can the Origin of the Genetic Code Be Explained by Direct RNA Templating?,” *BIO-Complexity*, Vol. 2011 (2).**

This peer-reviewed paper had its origins in a debate at Biola University in 2009 where Stephen Meyer debated two critical biologists. One of those scientists was Arthur Hunt from the University of Kentucky, who had previously cited the research of Michael Yarus which proposed that certain chemical affinities between RNA triplets and amino acids could have formed a chemical basis for the origin of the genetic code. According to Hunt, Yarus's research showed that “chemistry and physics ... can account for the origin of the genetic code” and thus “the very heart of Meyer's thesis (and his book [Signature in the Cell]) is wrong.” Meyer and Nelson's *BIO-Complexity* paper responds to Yarus's claims, showing that when challenged, ID proponents can produce compelling technical rebuttals. According to their detailed response, Yarus's (and Hunts') claims fail due to “selective use of data, incorrect null models, a weak signal even from positive results, ... and unsupported assumptions about the pre-biotic availability of amino acids.” Rather than refuting design, the research shows the need for “an intelligently-directed” origin of the code.

- **Ann K. Gauger and Douglas D. Axe, “The Evolutionary Accessibility of New Enzyme Functions: A Case Study from the Biotin Pathway,” *BIO-Complexity*, Vol. 2011 (1).**
This paper reports research conducted by Biologic Institute scientists Ann Gauger and Douglas Axe on the number of minimum changes that would be required to evolve one protein into another protein with a different function. The investigators studied two proteins, Kbl and BioF, with different functions but highly similar structures -- thought by evolutionists to be very closely related. Through mutational analysis, Gauger and Axe found that a minimum of *seven* independent mutations -- and probably many more -- would be necessary to convert Kbl to perform the function of its allegedly close genetic relative, BioF. Per Axe’s 2010 *BIO-Complexity* paper, “The Limits of Complex Adaptation: An Analysis Based on a Simple Model of Structured Bacterial Populations,” they report that this is beyond the limits of Darwinian evolution:

The extent to which Darwinian evolution can explain enzymatic innovation seems, on careful inspection, to be very limited. Large-scale innovations that result in new protein folds appear to be well outside its range. This paper argues that at least some small-scale innovations may also be beyond its reach. If studies of this kind continue to imply that this is typical rather than exceptional, then answers to the most interesting origins questions will probably remain elusive until the full range of explanatory alternatives is considered.

- **Ann K. Gauger, Stephanie Ebnet, Pamela F. Fahey, and Ralph Seelke, “Reductive Evolution Can Prevent Populations from Taking Simple Adaptive Paths to High Fitness,” *BIO-Complexity*, Vol. 2010 (2).**
This research, published by molecular biologist Ann Gauger of the Biologic Institute, Ralph Seelke at the University of Wisconsin Superior started by breaking a gene in the bacterium *Escherichia coli* required for synthesizing the amino acid tryptophan. When the gene was broken in just one place, random mutations in the bacteria’s genome were capable of “fixing” the gene. But when two mutations were required to restore function, Darwinian evolution could not do the job. Such results show that it is extremely unlikely for blind and unguided Darwinian processes to find rare amino-acid sequences that yield functional proteins. In essence, functional proteins are multi-mutation features in the extreme.
- **Michael J. Behe, “Experimental Evolution, Loss-of-Function Mutations, and ‘The First Rule of Adaptive Evolution,’” *The Quarterly Review of Biology*, Vol. 85(4):1-27 (December 2010).**
This peer-reviewed paper by Michael Behe in the journal *Quarterly Review of Biology* helps explain why we don’t observe the evolution of new protein functions. After reviewing many studies on bacterial and viral evolution, he concluded that most adaptations at the molecular level “are due to the loss or modification of a pre-existing molecular function.” In other words, since Darwinian evolution proceeds along the path of least resistance, Behe found that organisms are far more likely to evolve by a losing a biochemical function than by gaining one. He thus concluded that “the rate of

appearance of an adaptive mutation that would arise from the diminishment or elimination of the activity of a protein is expected to be 100-1000 times the rate of appearance of an adaptive mutation that requires specific changes to a gene.” If Behe is correct, then molecular evolution faces a severe problem. If a loss (or decrease) of function is much more likely than a gain-of-function, logic dictates that eventually an evolving population will run out of molecular functions to lose or diminish. Behe’s paper suggests that if Darwinian evolution is at work, something else must be generating the information for new molecular functions.

- **Douglas D. Axe, “The Limits of Complex Adaptation: An Analysis Based on a Simple Model of Structured Bacterial Populations,” *BIO-Complexity*, Vol. 2010 (4).**

The ability of Darwinian evolution to produce features that require multiple mutations before providing a benefit has been an issue long debated between proponents of intelligent design and proponents of neo-Darwinism. This paper responds to arguments from Michael Lynch and Adam Abegg, finding that they made a mistake -- actually two mistakes -- in their calculation of the length of time required for multiple mutations to occur when there is no adaptive benefit until all mutations are in place.

The purpose of Axe’s paper is then to mathematically determine how much time is needed to evolve traits that require multiple mutations before any adaptive benefit is conferred on the organism. He notes that there are essentially three models that might be invoked to explain the origin of these complex features: molecular saltation, sequential fixation, and stochastic tunneling. Axe’s paper tackles stochastic tunneling, a model that is in a sense midway between the molecular saltation and sequential fixation models. According to Axe, stochastic tunneling “differs from sequential fixation only in that it depends on each successive point mutation appearing without the prior one having become fixed.” However, because the prior mutations are not yet fixed in the larger population, this means that the number of organisms that have the prior mutations may be small. Thus, this mechanism “must instead rely on the necessary mutations appearing within much smaller subpopulations,” or as Axe models it, bacterial lines. This model resembles molecular saltation in that it depends on all required mutations eventually appearing by chance -- but anticipates this will happen after mutations are fixed in smaller subpopulations. Axe explains why all of these models face unavoidable statistical improbabilities: “in view of the fact that the underlying limitation is an unavoidable aspect of statistics -- that independent rare events only very rarely occur in combination -- it seems certain that all chance-based mechanisms must encounter it.”

Axe thus aims to accurately model the evolution of a multi-mutation feature. He investigates two cases: (1) when intermediate mutations are slightly disadvantageous, and (2) when intermediate mutations are selectively neutral. Axe seeks to give neo-Darwinian evolution a generous helping of probabilistic resources by modeling the evolution of bacteria -- asexual organisms that reproduce quickly and have very large effective population sizes. Unsurprisingly, Axe found that Darwinian evolution has great

difficulty fixing multiple mutations when those mutations have negative selection coefficients (i.e., they are disadvantageous, or maladaptive). Neutral mutations have a better shot at becoming fixed, but even here Axe finds that the ability of neo-Darwinian evolution to produce multi-mutation features is severely limited. The implications of this analysis for Darwinian evolution are large and negative. Axe's model made assumptions which were very generous towards Darwinian evolution. He assumed the existence of a huge population of asexually reproducing bacteria that could replicate quickly -- perhaps nearly three times per day -- over the course of billions of years. In these circumstances, complex adaptations requiring up to six mutations with neutral intermediates can become fixed. Beyond that, things become implausible. If only slightly maladaptive intermediate mutations are required for a complex adaptation, only a couple of mutations (at most two) could be fixed. If highly maladaptive mutations are required, the trait will never appear. Axe discusses the implications of his work:

In the end, the conclusion that complex adaptations cannot be very complex without running into feasibility problems appears to be robust. ... Although studies of this kind tend to be interpreted as supporting the Darwinian paradigm, the present study indicates otherwise, underscoring the importance of combining careful measurements with the appropriate population models.

Axe's paper, because it focuses on bacteria, does not model the evolution of sexually reproducing organisms. In sexually reproducing eukaryotic organisms, the longer generation times and lower effective population sizes would dramatically lower the number of mutations that could be fixed before acquiring some adaptive benefit. In vertebrate evolution, the probabilistic resources available to Darwinian evolution would be much smaller than those available to bacteria, and the result proportionately difficult to explain along Darwinian lines. Some other mechanism must be generating complex multi-mutation features.

- **Wolf-Ekkehard Lönnig, "Mutagenesis in *Physalis pubescens* L. ssp. *floridana*: Some further research on Dollo's Law and the Law of Recurrent Variation," *Floriculture and Ornamental Biotechnology*, 1-21 (2010).**

This original research paper on mutagenesis in plants favorably cites "intelligent design proponents," including Michael Behe, William Dembski, Jonathan Wells, and Stephen Meyer, as advocating one of various legitimate "scientific theories on the origin of species." Citing skeptics of neo-Darwinism such as Behe and "the almost 900 scientists of the Scientific Dissent from Darwinism," the paper notes that:

Many of these researchers also raise the question (among others), why -- even after inducing literally billions of induced mutations and (further) chromosome rearrangements -- all the important mutation breeding programs have come to an end in the Western world instead of eliciting a revolution in plant breeding, either by successive rounds of selective "micromutations" (cumulative selection in the sense of the modern synthesis), or by "larger mutations" ... and why the law of recurrent

variation is endlessly corroborated by the almost infinite repetition of the spectra of mutant phenotypes in each and any new extensive mutagenesis experiment (as predicted) instead of regularly producing a range of new systematic species...

Lönnig focuses on the origin of a particular trait found in some angiosperms, where longer sepals form a shelter for developing fruit called inflated calyx syndrome, or “ICS.” According to Lönnig, phylogenetic data indicate that under a neo-Darwinian interpretation, this trait was either lost in multiple lineages or evolved independently multiple times. If the trait evolved multiple times independently, then why do so many plants still lack such a “lantern” protective shelter? After noting that some proponents of neo-Darwinism make unfalsifiable appeals to unknown selective advantages, he concludes that neo-Darwinism is not making falsifiable predictions and finds that this “infinity of mostly non-testable explanations (often just-so-stories) itself may put the theory outside science.”

However, there is another possibility, namely the scientific hypothesis of intelligent design. In contrast to neo-Darwinism, the author notes the ID view can “be falsified by proving (among other points) that the probability to form an ICS by purely natural processes is high, that specified complexity is low, and finally, by generating an ICS by random mutations in a species displaying none.” Lönnig recounts the many phrases Darwin used to explain that his theory of evolution requires “innumerable slight variations,” and argues that the ICS could not evolve in such a stepwise fashion. After reviewing the multiple complex steps involved in forming an ICS, he states that his research “appears to be in agreement with Behe’s studies (2007): it seems to be very improbable that the current evolutionary theories like the modern synthesis (continuous evolution) or the hopeful monster approach (in one or very few steps) can satisfactorily explain the origin of the ICS.” In closing, Lönnig cites further Behe’s concept of irreducible complexity and Dembski’s arguments regarding the universal probability bound, contending that the ICS may be beyond the edge of evolution. Nevertheless, he leaves the present question open for further research, which he enthusiastically invites. Yet, citing the work of Stephen Meyer, William Dembski, and Robert Marks, he concludes that “it appears to be more than unlikely to generate the whole world of living organisms by the neo-Darwinian method.”

- **George Montañez, Winston Ewert, William A. Dembski, and Robert J. Marks II, “A Vivisection of the ev Computer Organism: Identifying Sources of Active Information,” *BIO-Complexity*, Vol. 2010 (3).**

This paper continues the work of the Evolutionary Informatics Lab showing that some cause other than Darwinian mechanisms is required to produce new information. Thomas Schneider’s “ev” program has been widely cited as showing that Darwinian processes can increase information. In this peer-reviewed paper, William Dembski and his coauthors demonstrate that, contrary to such claims, the “ev” program is in fact rigged to produce a particular outcome. According to the paper ev “exploit[s] one or more sources of knowledge to make the [evolutionary] search successful” and this

knowledge “predisposes the search towards its target.” They explain how the program smuggles in active information:

The success of ev is largely due to active information introduced by the Hamming oracle and from the perceptron structure. It is not due to the evolutionary algorithm used to perform the search. Indeed, other algorithms are shown to mine active information more efficiently from the knowledge sources provided by ev.

Schneider claims that ev demonstrates that naturally occurring genetic systems gain information by evolutionary processes and that “information gain can occur by punctuated equilibrium.” Our results show that, contrary to these claims, ev does not demonstrate “that biological information...can rapidly appear in genetic control systems subjected to replication, mutation, and selection.” We show this by demonstrating that there are at least five sources of active information in ev.

1. The perceptron structure. The perceptron structure is predisposed to generating strings of ones sprinkled by zeros or strings of zeros sprinkled by ones. Since the binding site target is mostly zeros with a few ones, there is a greater predisposition to generate the target than if it were, for example, a set of ones and zeros produced by the flipping of a fair coin.

2. The Hamming Oracle. When some offspring are correctly announced as more fit than others, external knowledge is being applied to the search and active information is introduced. As with the child’s game, we are being told with respect to the solution whether we are getting “colder” or “warmer.”

3. Repeated Queries. Two queries contain more information than one. Repeated queries can contribute active information.

4. Optimization by Mutation. This process discards mutations with low fitness and propagates those with high fitness. When the mutation rate is small, this process resembles a simple Markov birth process that converges to the target.

5. Degree of Mutation. As seen in Figure 3, the degree of mutation for ev must be tuned to a band of workable values.

A critic might claim that some of these items represent a proper modeling of Darwinian evolution. However, the way that ev uses these processes is unlike Darwinian evolution. For example, in (1), we see that the program’s use of a “perceptron” causes the output to be highly biased towards matching the target. It’s a way of cheating to ensure the program reaches its target sequence. Likewise, in (2) and (4), the program can effectively look ahead and march in the right direction towards the target, whereas unguided Darwinian evolution would have no “look ahead” capability. The active information in the Hamming Oracle makes a sharp contrast with the evolution of real

binding sites where there may be no binding capability until multiple mutations are fixed.

Mutation and selection are not the causes of success in these genetic algorithms. Yes, random mutation occurs and yes, there is selection. But selection is performed by a fitness function that is encoded by the programmer. And in programs like *ev*, the programmer intentionally shapes the fitness function to be amenable to stepwise Darwinian evolution. This effectively assumes the truth of Darwinian evolution. But in the real world of biology, fitness functions might look very different: there might be lonely islands of function in a vast sea of nonfunctional sequences. Indeed, if one uses a randomized fitness function, the search performs poorly and might not even outperform a blind search.

Thus choosing the right fitness function (from the set of possible fitness functions) requires as much or more information than choosing the right string from the set of possible strings in your search space. The fitness function itself is an information-rich structure. The program starts with this information-rich fitness function, and then produces something much less information rich -- the target sequence. And as the paper shows, *ev* does this in a relatively inefficient way: using the same information-rich fitness function, you can find the target 700 times more efficiently than by using simple single-agent stochastic hill climbing. Active information is smuggled into the fitness function. Rather than showing that information can arise by Darwinian evolution, *ev* shows that intelligence is required.

- **William A. Dembski and Robert J. Marks II, “The Search for a Search: Measuring the Information Cost of Higher Level Search,” *Journal of Advanced Computational Intelligence and Intelligent Informatics*, Vol. 14 (5):475-486 (2010).**
This paper by leading ID theorists William Dembski and Robert Marks argues that without information about a target, anything greater than a trivial search is bound to fail: “Needle-in-the-haystack problems look for small targets in large spaces. In such cases, blind search stands no hope of success.” They cite “No Free Lunch theorems,” according to which “any search technique will work, on average, as well as a blind search.” However, in such a case, “Success requires an assisted search. But whence the assistance required for a search to be successful?” Dembski and Marks thus argue that “successful searches do not emerge spontaneously but need themselves to be discovered via a search.” However, without information about the target, the search for a search itself is still no better than a blind search: “We prove two results: (1) The Horizontal No Free Lunch Theorem, which shows that average relative performance of searches never exceeds unassisted or blind searches, and (2) The Vertical No Free Lunch Theorem, which shows that the difficulty of searching for a successful search increases exponentially with respect to the minimum allowable active information being sought.” The implication, of course, is that without the ultimate input from an intelligent agent -- active information -- such searches will fail.

- **Douglas D. Axe, “The Case Against a Darwinian Origin of Protein Folds,” *BIO-Complexity*, Vol. 2010 (1).**

This paper by Biologic Institute director Douglas Axe argues that amino-acid sequences that produce functional protein folds are too rare to be discovered by the trial-and-error processes of Darwinian evolution. It begins by observing that when the genetic code was first discovered, “The code had made it clear that the vast set of possible proteins that could conceivably be constructed by genetic mutations is far too large to have actually been sampled to any significant extent in the history of life. Yet how could the highly incomplete sampling that has occurred have been so successful? How could it have located the impressive array of protein functions required for life in all its forms, or the comparably impressive array of protein structures that perform those functions? This concern was raised repeatedly in the early days of the genetic code, but it received little attention from the biological community.” After reviewing the problem, Axe concludes that “With no discernable shortcut to new protein folds, we conclude that the sampling problem really is a problem for evolutionary accounts of their origins.” He argues that “a search mechanism unable to locate a small patch on a grain of level-14 sand is not apt to provide the explanation of fold origins that we seek. Clearly, if this conclusion is correct it calls for a serious rethink of how we explain protein origins, and that means a rethink of biological origins as a whole.”

- **Winston Ewert, George Montañez, William Dembski and Robert J. Marks II, “Efficient Per Query Information Extraction from a Hamming Oracle,” *42nd South Eastern Symposium on System Theory*, pp. 290-297 (March, 2010).**

This paper continues the peer-reviewed work co-published by William Dembski, Robert Marks, and others affiliated with the Evolutionary Informatics Lab. Here, the authors argue that Richard Dawkins’s “METHINKSITISLIKEAWASEL” evolutionary algorithm starts off with large amounts of active information -- that is, information intelligently inserted by the programmer to aid the search. This paper covers all of the known claims of operation of the WEASEL algorithm and shows that in all cases, active information is used. Dawkins’s algorithm can best be understood as using a “Hamming oracle” as follows: “When a sequence of letters is presented to a Hamming oracle, the oracle responds with the Hamming distance equal to the number of letter mismatches in the sequence.” The authors find that this form of a search is very efficient at finding its target -- but that is only because it is preprogrammed with large amounts of active information needed to quickly find the target. This preprogrammed active information makes it far removed from a true Darwinian evolutionary search algorithm. An online toolkit of programs called “Weasel Ware” accompanies the paper and can be found at <http://evoinfo.org/weasel>.

- **David L. Abel, “Constraints vs Controls,” *The Open Cybernetics and Systemics Journal*, Vol. 4:14-27 (January 20, 2010).**

This article explains that the organization of matter in life requires non-material causes such as “mental choice of tokens (physical symbol vehicles) in a material symbol system” which then “instantiates non-physical formal Prescriptive Information (PI) into

physicality.” It also acknowledges that life is fundamentally based upon information: “Life, on the other hand, is highly informational. Metabolic organization and control is highly programmed. Life is marked by the integration of large numbers of computational solutions into one holistic metasystem. No as-of-yet undiscovered law will ever be able to explain the highly informational organization of living organisms.” The article explains that “choice contingency” is a concept where the outcome is determined by the choice of an intelligent agent:

Whereas chance contingency cannot cause any physical effects, choice contingency can. But choice contingency, like chance contingency, is formal, not physical. So how could non-physical choice contingency possibly become a cause of physical effects? The answer lies in our ability to instantiate formal choices into physical media. As we shall see below, formal choices can be represented and recorded into physicality using purposefully chosen physical symbol vehicles in an arbitrarily assigned material symbol system. Choices can also be recorded through the setting of configurable switches. Configurable switches are physiodynamically indeterminate (inert; decoupled from and incoherent with physiodynamic causation). This means that physiodynamics plays no role in how the switch is set. Physiodynamic factors are equal in the flipping of a binary switch regardless of which option is formally chosen. Configurable switches represent decision nodes and logic gates. They are set according to arbitrary rules, not laws. Here arbitrary does not mean random. Arbitrary means “not physiodynamically determined.” Rules are not constrained by physical nature. Arbitrary means “freely selectable” -- choice contingent.

Only an intelligent cause -- an “agent” -- could implement such choice contingency. The article further explains that physical constraints are not what govern life, but rather choice controls, which cannot be explained by metaphysical naturalism:

Volition (choice contingency) is every bit as repeatedly observable, predictable (given any form of true organization), and as potentially falsifiable as any naturalistic hypothesis. Volition and control are no more metaphysical than acceleration, wave/particle duality, weight, height, quarks, and light. We cannot label volition and control “metaphysical,” and quantum mechanics and statistical mechanics “physical.” Mathematics and the scientific method themselves are non-physical. Volitional controls (as opposed to mere constraints) are a fact of objective reality. If this fact does not fit within the perimeter of our prized lifelong worldview, perhaps it is time to open our minds and reconsider the purely metaphysical presuppositions that shaped that inadequate worldview. Philosophic naturalism cannot empirically or logically generate organizational bona fide controls. It can only generate self-ordering, low-informational, unimaginative constraints with no formal cybernetic capabilities. Metaphysical naturalism is too small a perimeter to contain all of the pieces. Naturalism is too inadequate a metanarrative to be able to incorporate all of the observable scientific data.

The article concludes that the formalisms we see in life “arise only in the minds of agents.”

- **David L. Abel, “The GS (genetic selection) Principle,” *Frontiers in Bioscience*, Vol. 14:2959-2969 (January 1, 2010).**

This paper studies the genetic code, observing that “Nucleotides function as physical symbol vehicles in a material symbol system.” But it argues that teleology is necessary to explain the choice controls in such systems: “The challenge of finding a natural mechanism for linear digital programming extends from primordial genetics into the much larger realm of semantics and semiotics in general. Says Barham: ‘The main challenge for information science is to naturalize the semantic content of information. This can only be achieved in the context of a naturalized teleology (by ‘teleology’ is meant the coherence and the coordination of the physical forces which constitute the living state)’. The alternative term ‘teleonomy’ has been used to attribute to natural process ‘the appearance of teleology’. Either way, the bottom line of such phenomena is selection for higher function at the logic gate programming level.” The article explains why natural selection is inadequate to explain many features we observe in biology, and why instead we require a cause that can anticipate function: “Programming selections at successive decision nodes requires anticipation of what selections and what sequences would be functional. Selection must be for potential function. Nature cannot anticipate, let alone plan or pursue formal function. Natural selection can only preserve the fittest already-existing holistic life.”

- **D. Halsmer, J. Asper, N. Roman, and T. Todd, “The Coherence of an Engineered World,” *International Journal of Design & Nature and Ecodynamics*, Vol. 4(1):47–65 (2009).**

This peer-reviewed scientific paper argues that we live in an “engineered world.” It observes that “Human-engineered systems are characterized by stability, predictability, reliability, transparency, controllability, efficiency, and (ideally) optimality. These features are also prevalent throughout the natural systems that make up the cosmos. However, the level of engineering appears to be far above and beyond, or transcendent of, current human capabilities.” The paper cites the fine-tuning of the universe for life, such as the special properties of water, the prevalence of elements needed for life (e.g. hydrogen, oxygen, and carbon), the expansion rate of the universe, as well as the Galactic Habitable Zone, a concept developed by Discovery Institute senior fellow Guillermo Gonzalez:

On the universal scale, however, one can see that our planet is in a comparatively narrow region of space known as the “Galactic Habitable Zone.” This zone allows for the right surface temperature, stable climate metallicity, ability to hold liquid water, and many other conditions necessary for life. There is no practical reason why the universe has to contain life, but the fact that it does gives great importance to this zone for the benefit of our existence.

The authors then explain Gonzalez and Jay Richards's "Privileged Planet" argument:

Not only does this zone satisfy the requirements of life but also it endowed humans with a prime position to view the wonders of the universe. There are many qualities that make the earth an excellent place from which to study the universe. First of all is the transparency of the atmosphere. Our atmosphere admits the radiation necessary for life while blocking most of its lethal energy. This transparency also allows humans to see into space without the distortions caused by a thick atmosphere as would be the case on Venus. Secondly, the regularity of our solar system's orbits makes time calculation of planetary events more predictable, even allowing for estimations of planetary orbits millions of years ago. Finally, the gas and dust in our region of the Milky Way are diffuse compared to other regions in the local mid-plane. This allows humans to view 80% of the universe without blockage. If our solar system was moved farther away, perpendicularly to the mid-plane, we would be able to see the other 20%. However, this would cause a large percentage of our current view to be blocked by dust as well as the luminosity of stars in close proximity. Humanity's place in the universe is amazingly unique when it comes to discovery. Planet earth is in prime position for the gleaning of knowledge from the stars.

The paper also focuses on fine-tuning in biology as evidence of biological design, citing the work of a variety of noteworthy proponents of intelligent design, including Walter Bradley, Michael Behe, Jonathan Wells, and William Dembski. The paper examines the engineering of life, noting that "[b]iological systems are constantly undergoing processes that exhibit modularity, specificity, adaptability, durability, and many other aspects of engineered systems." It quotes from William Dembski and Jonathan Wells's book *The Design of Life*, stating: "Many of the systems inside the cell represent nanotechnology at a scale and sophistication that dwarfs human engineering. Moreover, our ability to understand the structure and function of these systems depends directly on our facility with engineering principles." The authors further cite the work of Michael Behe's, such as *Darwin's Black Box* and *The Edge of Evolution*, explaining that biological systems display "irreducible complexity" which requires a goal-directed process or "'bottom up-top down' design." After examining the engineering of our universe from the macro- to microscope scales, they conclude: "An interdisciplinary study of the cosmos suggests that a transcendently engineered world may be the most coherent explanation for the reality we experience as human beings."

- **Ossi Turunen, Ralph Seelke, and Jed Macosko, "In silico evidence for functional specialization after genome duplication in yeast," *Federation of European Microbiological Societies (FEMS) Yeast Research*, Vol. 9: 16-31 (2009).**

This paper by ID-friendly biologists discusses the evidence for gene duplication in yeast, and the implications for Darwinian evolution. They argue that when gene duplicates acquire "new" functions, it's not because a "new" function evolved, but rather because the original gene had multiple functions, some of which are then lost, as the gene

becomes more “specialized.” Called the promiscuous model of protein evolution, it proposes that examples of evolution by gene duplication results more from loss, rather than gain, of function:

[T]here appears to be a trend that the complexity of the genes (amount of functions in one gene) is slowly decreasing due to gene duplication and subsequent divergence. Functional reduction of the fast-evolving genes in the duplicated gene pairs is also seen in the finding that they have less protein-protein interactions.

This could have important implications for Darwinian evolution, since a mechanism that tends to reduce complexity and protein-protein interactions obviously cannot account for the ultimate origin of genes.

- **Richard A. Carnhart and Adam Cenian, “Implication of Proven Limits on Scientific Knowledge: Gödel’s Proof, Quantum Uncertainty, Chaos Theory and Specified Complexity of Information Theory,” *Bulletin de la Société Des Sciences Et Des Lettres de Łódź*, Vol. LIX (Série: Recherches Sur Les Déformations LVIII): 7-18 (2009).**

This article in a French scientific journal argues that “four discoveries of modern science: Gödel’s incompleteness theorems, quantum uncertainty, chaos theory, and, tentatively, complex specified information theory, show us specific ways in which our ability to know and control nature is limited in principle, not only in practice.” After observing that the scientism of atheists like Peter Atkins “rules out the idea that the universe was created for a purpose or was designed,” they argue that other factors lead to the opposite conclusion. In particular, one factor they cite is “specified complexity”:

Even organisms made of a single cell have a large genetic code in the form of a base 4 coding system (using the letters A., C., G. T). This genetic code is like a computer program. An arbitrary single sequence of a million of these letters is extremely improbable. Further, the vast majority of such sequences would not allow an organism to form, much less function. Only very specific sequences can code for the processes in the organism’s living cell. These processes are many, highly complex, and highly coordinated with one another. The term “specified complexity” has been suggested to describe such living systems.

But where does specified complexity come from? According to the paper, “In the entire experience of the human race apart from living systems, no such specified complex systems, very rich in information, ever raise except as the result of design and action of an intelligent source.” They argue that “it may be necessary to postulate action of an intelligence inside or outside of the natural order to explain the origin of complex specified information (CSI) in living systems.” The authors recognize, however, that the scientific community is closed to this conclusion, and urges them to keep an open mind:

It seems that the scientific community may be so comfortable with Neo-Darwinism that it has not invested in the scientific development of life-related information

theory. This is a genuine issue of freedom of inquiry in intellectual life, whether in the university or the academy. This author recommends: give free inquiry a chance!

The authors conclude that the evidence suggests “a more encompassing world view than mere ontological naturalism.”

- **Winston Ewert, William A. Dembski, and Robert J. Marks II, “Evolutionary Synthesis of Nand Logic: Dissecting a Digital Organism,” *Proceedings of the 2009 IEEE International Conference on Systems, Man, and Cybernetics*, pp. 3047-3053 (October, 2009).**

In 2003, researchers at the University of Michigan published in *Nature* the results of a computer simulation of evolution called Avida. The *Nature* paper’s authors expressly framed “Avida” as a refutation of ID arguments, claiming the program shows “that complex adaptive traits do emerge via standard Darwinian mechanisms.” But does Avida truly model “standard Darwinian mechanisms”? In 2009, four researchers at the pro-ID Evolutionary Informatics Lab furthered this scientific debate in a peer-reviewed paper titled, “Evolutionary Synthesis of Nand Logic: Dissecting a Digital Organism.” Building upon concepts previously established by William Dembski and Robert J. Marks, the paper argues that Avida’s programmers smuggle in “active information” to allow their simulation to find its evolutionary targets. According to the paper, sources of intelligently programmed “active information” in Avida include the following:

- “Active information from Avida’s initialization” where “[t]he initialization in Avida recognizes the essential role of the nop-C instruction in finding the EQU.”
- “Mutation, fitness, and choosing the fittest of a number of mutated offspring.”
- Most importantly, there is “Stair step active information” where the digital “mutations” in Avida are essentially pre-programmed to perform a useful function, and are rewarded for doing so.

Ewert, Dembski, and Marks focus on this third point, noting that, “The importance of stair step active information is evident from the inability to generate a single EQU [the target function] in Avida without using it.” They ask, “What happens when no stair step active information is applied?” and note what the original authors of the Avida paper in *Nature* reveal:

At the other extreme, 50 populations evolved in an environment where only EQU was rewarded, and no simpler function yielded energy. We expected that EQU would evolve much less often because selection would not preserve the simpler functions that provide foundations to build more complex features. Indeed, none of these populations evolved EQU, a highly significant difference from the fraction that did so in the reward-all environment.

But does real biology “reward” mutations to the extent that Avida does? The passage quoted above shows that when Avida is calibrated to model actual biology -- where many changes may be necessary before there is any beneficial function to select for

(irreducible complexity) -- “none of these populations evolved” the target function. Avida’s creators trumpet its success, but Ewert, Dembski, and Marks show that Avida uses “stair step active information” by rewarding forms of digital “mutations” that are pre-programmed to yield the desired outcome. It does not model true Darwinian evolution, which is blind to future outcomes and cannot use active information. The implications may be unsettling for proponents of neo-Darwinian theory: Not only is Darwinian evolution “on average... no better than blind search,” but Avida is rigged by its programmers to succeed, showing that intelligence is in fact necessary to generate complex biological features. An online toolkit of programs called “Mini Vida” accompanies the paper and can be found at <http://evoinfo.org/minivida>.

- **William A. Dembski and Robert J. Marks II, “Bernoulli’s Principle of Insufficient Reason and Conservation of Information in Computer Search,” *Proceedings of the 2009 IEEE International Conference on Systems, Man, and Cybernetics*, pp. 2647 – 2652 (October, 2009).**

In his 2001 book *No Free Lunch*, William Dembski argued that Darwinian evolutionary searches cannot produce new complex and specified information, and information that is “found” by Darwinian searches actually reflects information that was smuggled in by an intelligence external to the search. This peer-reviewed paper co-written with Robert J. Marks furthers Dembski’s arguments, contending that in all searches -- including Darwinian ones -- information is conserved such that “on average no search outperforms any other.” The implication of their principle of “Conservation of Information” (COI) is that Darwinian evolution, at base, is actually no better than a random search. To make their argument, the paper develops a methodology for measuring the information smuggled into a search algorithm by intelligence. Exogenous Information (I_{Ω}) represents the difficulty a search in finding its target with no prior information about its location. Active Information (I_{+}) is the amount of information smuggled in by intelligence to aid the search algorithm in finding its target. Endogenous Information (I_{S}) then measures the difficulty the search will have in finding its target after the addition of Active Information. Thus, $I_{+} = I_{\Omega} - I_{S}$. Having laid this theoretical groundwork, Dembski and Marks begin to apply their ideas to evolutionary algorithms which claim to produce new information. They argue that computer simulations often do not properly model truly unguided Darwinian evolution: “COI has led to the formulation of active information as a measure that needs to be introduced to render an evolutionary search successful. Like an athlete on steroids, many such programs are doctored, intentionally or not, to succeed,” and thus “COI puts to rest the inflated claims for the information generating power of evolutionary simulations such as Avida and ev.” They conclude that when trying to generate new complex and specified information, “in biology, as in computing, there is no free lunch,” and therefore some assistance from intelligence is required to aid Darwinian evolution find unlikely targets in search space.

- **William A. Dembski and Robert J. Marks II, “Conservation of Information in Search: Measuring the Cost of Success,” *IEEE Transactions on Systems, Man, and Cybernetics-Part A: Systems and Humans*, Vol. 39(5):1051-1061 (September, 2009).**

This peer-reviewed article by William A. Dembski and Robert J. Marks II challenges the ability of Darwinian processes to create new functional genetic information. Darwinian evolution is, at its heart, a search algorithm that uses a trial and error process of random mutation and unguided natural selection to find genotypes (i.e., DNA sequences) that lead to phenotypes (i.e., biomolecules and body plans) that have high fitness (i.e., foster survival and reproduction). Dembski and Marks's article explains that unless you start with some information about where peaks in a fitness landscape may lie, any search -- including Darwinian searches -- are on average no better than a random search. After assessing various examples of evolutionary searches, Dembski and Marks show that attempts to model Darwinian evolution via computer simulations, such as Richard Dawkins' famous "METHINKSITISLIKEAWEASEL" exercise, start off with, as Dembski and Marks put it, "problem-specific information about the search target or the search-space structure." According to the paper, such simulations only reach their evolutionary targets because there is pre-specified "accurate information to guide them," or what they call "active information." The implication, of course, is that some intelligent programmer is required to front-load a search with active information if the search is to successfully find rare functional genetic sequences. They conclude that "Active information is clearly required in even modestly sized searches." This paper is in many ways a validation of some of Dembski's core ideas in his 2001 book *No Free Lunch: Why Specified Complexity Cannot Be Purchased without Intelligence*, which argued that some intelligent input is required to produce novel complex and specified information. Dembski has [written](#) about this article at Uncommon Descent, explaining how it supports ID: "Our critics will immediately say that this really isn't a pro-ID article but that it's about something else (I've seen this line now for over a decade once work on ID started encroaching into peer-review territory). Before you believe this, have a look at the article. In it we critique, for instance, Richard Dawkins' METHINKS*IT*IS*LIKE*A*WEASEL (p. 1055). Question: When Dawkins introduced this example, was he arguing pro-Darwinism? Yes he was. In critiquing his example and arguing that information is not created by unguided evolutionary processes, we are indeed making an argument that supports ID."

- **David L. Abel, "The Universal Plausibility Metric (UPM) & Principle (UPP)," *Theoretical Biology and Medical Modelling*, Vol. 6(27) (2009).**

Materialists often vaguely appeal to vast periods of time and boundless probabilistic resources in the universe to make their scenarios sound plausible. But is "mere possibility" sufficient justification to assert "scientific plausibility"? This peer-reviewed article in *Theoretical Biology and Medical Modelling* answers that question, arguing that "[m]ere possibility is not an adequate basis for asserting scientific plausibility" because "[a] precisely defined universal bound is needed beyond which the assertion of plausibility, particularly in life-origin models, can be considered operationally falsified." The paper observes that "Combinatorial imaginings and hypothetical scenarios can be endlessly argued simply on the grounds that they are theoretically possible," but then argues that the unwillingness of materialists to consider certain origin of life models to be false is actually stopping the progress of science, since "at some point our reluctance to exclude any possibility becomes stultifying to operational science." The paper

observes that “Just because a hypothesis is possible should not grant that hypothesis scientific respectability,” an important rejoinder to materialists who propose speculative stories about self-organization or co-option to explain the origin of biological complexity. The author then rigorously calculates the Universal Plausibility Metric (UPM), incorporating the maximum probabilistic resources available for the universe, galaxy, solar system, and the earth:

$$\begin{aligned} {}^c\Omega_u &= \text{Universe} = 10^{13} \text{ reactions/sec} \times 10^{17} \text{ secs} \times 10^{78} \text{ atoms} = 10^{108} \\ {}^c\Omega_g &= \text{Galaxy} = 10^{13} \times 10^{17} \times 10^{66} = 10^{96} \\ {}^c\Omega_s &= \text{Solar System} = 10^{13} \times 10^{17} \times 10^{55} = 10^{85} \\ {}^c\Omega_e &= \text{Earth} = 10^{13} \times 10^{17} \times 10^{40} = 10^{70} \end{aligned}$$

The author concludes that consideration of Universal Plausibility Metrics allow for falsification of speculative origin of life scenarios: “The application of The Universal Plausibility Principle (UPP) precludes the inclusion in scientific literature of wild metaphysical conjectures that conveniently ignore or illegitimately inflate probabilistic resources to beyond the limits of observational science.” When hypotheses require probabilistic resources that exceed these metrics, the author argues that they “should be considered not only operationally falsified hypotheses, but bad metaphysics on a plane equivalent to blind faith and superstition.” It concludes that the complexity we see in life requires an agent-based cause that can make choices: “Symbol systems and configurable switch-settings can only be programmed with choice contingency, not chance contingency or fixed law, if non-trivial coordination and formal organization are expected.”

- **David L. Abel, “The Capabilities of Chaos and Complexity,” *International Journal of Molecular Sciences*, Vol. 10:247-291 (2009).**

This paper seeks to address the question, “If all known life depends upon genetic instructions, how was the first linear digital prescriptive genetic information generated by natural process?” The author warns materialists that there is an easy solution to the challenges posed by intelligent design: “To stem the growing swell of Intelligent Design intrusions, it is imperative that we provide *stand-alone natural process* evidence of non-trivial self-organization at the edge of chaos. We must demonstrate on sound scientific grounds the formal capabilities of naturally occurring physicydynamic complexity.” However, while the author notes that much effort has been spent “arguing to the lay community that we *have proved* the current biological paradigm,” he concludes that the actual evidence for self-organization is “sorely lacking” and has been “inflated.” The author emphasizes a distinction between “order” and “organization,” arguing that self-ordered structures like whirlpools are readily constructed by natural processes, but “have never been observed to achieve 1) programming, 2) computational halting, 3) creative engineering, 4) symbol systems, 5) language, or 6) bona fide organization” -- all hallmarks of living organisms. In contrast, living organisms are built upon programming and are highly organized, but “physicydynamics alone cannot organize itself into formally functional systems requiring algorithmic optimization, computational halting,

and circuit integration.” His solution offers a positive argument for design: “No known natural process exists that spontaneously writes meaningful or functional syntax. Only agents have been known to write meaningful and pragmatic syntax.” He notes that the kind of “sophisticated formal function” found in life “consistently requires regulation and control,” but “Control always emanates from choice contingency and intentionality, not from spontaneous molecular chaos.”

- **David L. Abel, “The biosemiosis of prescriptive information,” *Semiotica*, Vol. 174(1/4):1-19 (2009).**

This article explains that classical measures of information, such as Shannon Information, are inadequate to explain biological function, suggesting that functional biological information be measured as prescriptive information (“PI”). It argues that the choice of an intelligent agent is necessary to produce PI: “PI arises from expedient choice commitments at bona fide decision nodes. Such decisions steer events toward pragmatic results that are valued by agents. Empirical evidence of PI arising spontaneously from inanimate nature is sorely lacking. Neither chance nor necessity has been shown to generate prescriptive information. Choice contingency, not chance contingency, prescribes non-trivial function.” According to the article, agent choice is required to generate the formalisms found in living organism: “Formalisms of all kinds involve abstract ideas and agent-mediated purposeful choices. Inanimate physics and chemistry have never been shown to generate life or formal choice-based systems.”

- **A. C. McIntosh, “Information and Entropy—Top-Down or Bottom-Up Development in Living Systems,” *International Journal of Design & Nature and Ecodynamics*, Vol. 4(4):351-385 (2009).**

This paper expressly endorses intelligent design after exploring a key question in ID thinking. “The ultimate question in origins must be: Can information increase in a purely materialistic or naturalistic way? It is not satisfactory to simply assume that information has to have arisen in this way. The alternative of original design must be allowed and all options examined carefully.” A professor of thermodynamics and combustion theory, McIntosh is well acquainted with the workings of machinery. His argument is essentially twofold: (1) First, he defines the term “machine” (a device which locally raises the free energy) and observes that the cell is full of machines. Such machines pose a challenge to neo-Darwinian evolution due to their irreducibly complex nature. (2) Second, he argues that the information in living systems (similar to computer software) uses such machines and in fact requires machines to operate (what good is a program without a computer to run it?). An example is the information in DNA. From a thermodynamics perspective, the only way to make sense of this is to understand that the information is non-material and constrains the thermodynamics so that the local matter and energy are in a non-equilibrium state. McIntosh addresses the objection that, thermodynamically speaking, highly organized low entropy structures can be formed at the expense of an increase in entropy elsewhere in the universe. However, he notes that this argument fails when applied to the origin of biological information:

whilst this argument works for structures such as snowflakes that are formed by natural forces, it does not work for genetic information because the information system is composed of machinery which requires precise and non-spontaneous raised free energy levels -- and crystals like snowflakes have zero free energy as the phase transition occurs.

McIntosh then tackles the predominant reductionist view of biological information which “regards the coding and language of DNA as essentially a phenomenon of the physics and chemistry of the nucleotides themselves.” He argues that this classical view is wrong, for “biological structures contain coded instructions which ... are not defined by the matter and energy of the molecules carrying this information.” According to McIntosh, Shannon information is not a good measure of biological information since it is “largely not relevant to functional information at the phenotype level.” In his view, “[t]o consider biological information as simply a 'by product' of natural selective forces operating on random mutations is not only counter-intuitive, but scientifically wrong.” According to McIntosh, one major reason for this is “the irreducibly complex nature of the machinery involved in creating the DNA/mRNA/ribosome/amino acid/protein/DNA-polymerase connections.” He continues:

All of these functioning parts are needed to make the basic forms of living cells to work. ... This, it may be argued, is a repeat of the irreducible complexity argument of Behe, and many think that that debate has been settled by the work of Pallen and Matzke where an attempt to explain the origin of the bacterial flagellum rotary motor as a development of the Type 3 secretory system has been made. However, this argument is not robust simply because it is evident that there are features of both mechanisms which are clearly not within the genetic framework of the other. That is, the evidence, far from pointing to one being the ancestor of the other, actually points to them both being irreducibly complex. In the view of the author this argument is still a very powerful one.

Further citing *Signature in the Cell*, McIntosh states: “What is evident is that the initial information content in DNA and living proteins rather than being small must in fact be large, and is in fact vital for any process to work to begin with. The issue of functional complexity and information is considered exhaustively by Meyer who argues that the neo-Darwinist model cannot explain all the appearances of design in biology.” So how do biological systems achieve their highly ordered, low-entropy states? McIntosh’s argument is complementary to that of Stephen Meyer’s, but it takes a more thermodynamic approach. According to McIntosh, information is what allows biological systems to attain their high degrees of order: “the presence of information is the cause of lowered logical entropy in a given system, rather than the consequence. In living systems the principle is always that the information is transcendent to, but *using raised free energy* chemical bonding sites.” McIntosh solves the problem of the origin of information by arguing that it must arise in a “top-down” fashion requiring the input of intelligence:

[T]here is a perfectly consistent view which is a top-down approach where biological information already present in the phenotypic creature (and not emergent as claimed in the traditional bottom-up approach) constrains the system of matter and energy constituting the living entity to follow intricate non-equilibrium chemical pathways. These pathways whilst obeying all the laws of thermodynamics are constantly supporting the coded software which is present within ... Without the addition of outside intelligence, raw matter and energy will not produce auto-organization and machinery. This latter assertion is actually repeatedly borne out by experimental observation -- new machinery requires intelligence. And intelligence in biological systems is from the non-material instructions of DNA.

This thinking can be applied to DNA: since “the basic coding is the cause (and thus reflects an initial purpose) rather than the consequence, [the top-down approach] gives a much better paradigm for understanding the molecular machinery which is now consistent with known thermodynamic principles.” McIntosh explains that the low-entropy state of biological systems is the result of the workings of machines, which must be built by intelligence: “It has often been asserted that the logical entropy of a non-isolated system could reduce, and thereby new information could occur at the expense of increasing entropy elsewhere, and without the involvement of intelligence. In this paper, we have sought to refute this claim on the basis that this is not a sufficient condition to achieve a rise in local order. One always needs a machine in place to make use of an influx of new energy and a new machine inevitably involves the systematic raising of free energies for such machines to work. Intelligence is a pre-requisite.” He concludes his paper with an express endorsement of intelligent design: “the implication of this paper is that it supports the so-called intelligent design thesis -- that an intelligent designer is needed to put the information into the biological system.”

- **A.C. McIntosh, “Evidence of design in bird feathers and avian respiration,” *International Journal of Design & Nature and Ecodynamics*, Vol. 4(2):154–169 (2009).** In this peer-reviewed paper, Leeds University professor Andy McIntosh argues that two systems vital to bird flight -- feathers and the avian respiratory system -- exhibit “irreducible complexity.” The paper describes these systems using the exact sort of definitions that Michael Behe uses to describe irreducible complexity:

[F]unctional systems, in order to operate as working machines, must have all the required parts in place in order to be effective. If one part is missing, then the whole system is useless. The inference of design is the most natural step when presented with evidence such as in this paper, that is evidence concerning avian feathers and respiration.

Regarding the structure of feathers, he argues that they require many features to be present in order to properly function and allow flight:

[I]t is not sufficient to simply have barbules to appear from the barbs but that opposing barbules must have opposite characteristics -- that is, hooks on one side of the barb and ridges on the other so that adjacent barbs become attached by hooked barbules from one barb attaching themselves to ridged barbules from the next barb (Fig. 4). It may well be that as Yu *et al.* suggested, a critical protein is indeed present in such living systems (birds) which have feathers in order to form feather branching, but that does not solve the arrangement issue concerning left-handed and right-handed barbules. It is that vital network of barbules which is necessarily a function of the encoded information (software) in the genes. Functional information is vital to such systems.

He further notes that many evolutionary authors “look for evidence that true feathers developed first in small non-flying dinosaurs before the advent of flight, possibly as a means of increasing insulation for the warm-blooded species that were emerging.” However, he finds that when it comes to fossil evidence for the evolution of feathers, “None of the fossil evidence shows any evidence of such transitions.”

Regarding the avian respiratory system, McIntosh contends that a functional transition from a purported reptilian respiratory system to the avian design would lead to non-functional intermediate stages. He quotes John Ruben stating, “The earliest stages in the derivation of the avian abdominal air sac system from a diaphragm-ventilating ancestor would have necessitated selection for a diaphragmatic hernia in taxa transitional between theropods and birds. Such a debilitating condition would have immediately compromised the entire pulmonary ventilatory apparatus and seems unlikely to have been of any selective advantage.” With such unique constraints in mind, McIntosh argues that “even if one does take the fossil evidence as the record of development, the evidence is in fact much more consistent with an *ab initio* design position -- that the breathing mechanism of birds is in fact the product of intelligent design.”

McIntosh’s paper argues that science must remain at least open to the possibility of detecting design in nature, since “to deny the possibility of the involvement of external intelligence is effectively an assumption in the religious category.” Since feathers and the avian respiratory system exhibit irreducible complexity, he expressly argues that science must consider the design hypothesis:

As examples of irreducible complexity, they show that natural systems have intricate machinery which does not arise in a “bottom up” approach, whereby some natural selective method of gaining small-scale changes could give the intermediary creature some advantage. This will not work since, first, there is no advantage unless all the parts of the new machine are available together and, second, in the case of the avian lung the intermediary creature would not be able to breathe, and there is little selective advantage if the creature is no longer alive. As stated in the introduction, the possibility of an intelligent cause is both a valid scientific assumption, and borne out by the evidence itself.

- **David L. Abel, “The ‘Cybernetic Cut’: Progressing from Description to Prescription in Systems Theory,”** *The Open Cybernetics and Systemics Journal*, Vol. 2:252-262 (2008). This article tries to explain how scientists can produce artificial intelligence and bridge the “cybernetic cut” -- from programmed reactions to real choices. It thus states: “How did inanimate nature give rise to an algorithmically organized, semiotic and cybernetic life? Both the practice of physics and life itself require traversing not only an epistemic cut, but a Cybernetic Cut. A fundamental dichotomy of reality is delineated. The dynamics of physicality (‘chance and necessity’) lie on one side. On the other side lies the ability to choose with intent what aspects of ontological being will be preferred, pursued, selected, rearranged, integrated, organized, preserved, and used (cybernetic formalism).” The article contends that choice contingency is necessary to produce functional biological life forms, for: “Choice contingency, on the other hand, involves purposeful selection from among real options. Unlike chance contingency, with choice contingency an internalized goal motivates each selection.” The paper further notes that “The capabilities of chance contingency are often greatly inflated,” suggesting that “agent steerage” is necessary to explain biological features. According to the paper “Purposeful choices are needed to achieve sophisticated formal utility. The chance and/or necessity of physiodynamics alone have never been observed to generate a nontrivial formal control system.”
- **Richard v. Sternberg, “DNA Codes and Information: Formal Structures and Relational Causes,”** *Acta Biotheoretica*, Vol. 56(3):205-232 (September, 2008). This article by pro-ID evolutionary biologist Richard Sternberg compares the information processing ability of the cell to computer programming. Sternberg observes that non-physical symbols and codes underlie biology, stating that “There are no chemical constraints or laws that explain the 64-to-20 mapping of codons to amino acids and stop sites -- the relations are ‘arbitrary’ with respect to the molecular components in the sense that mappings can be reassigned.” According to Sternberg, the genetic code is like computer codes in that it contains the following properties: “Redundancy, Error dampening capability, Symbolic and semantic flexibility, Output versatility, Multiple realizability, and Text editing.” There is also a computer-like form of recursivity in molecular biology, “as a protein product can in turn be part of the transcriptional, RNA processing, or translational apparatus -- even binding to its own mRNA.” He explains the interdependent nature of DNA and other biomolecules, stating “Any DNA code is but the domain of a larger system; the larger system in turn depends on DNA codes (at least in part).” The author’s conclusion is that the workings of biology, fundamentally, are not reducible to material molecules but rather resides in information, symbols, and sets of mathematically logical rules: “The mathematical structures that proteins (and RNAs!) are the result of are not ‘in’ a gene. Instead, the DNA sequence is the material platform for the symbol strings that allow information to be accessed. In this sense, then, DNA is less than its Central Dogma interpretation because it is not ontically informational. Yet DNA enables many more code systems tha[n] commonly acknowledged and in this way is more than just a collection of codons.”

- **Douglas D. Axe, Brendan W. Dixon, Philip Lu, “Stylus: A System for Evolutionary Experimentation Based on a Protein/Proteome Model with Non-Arbitrary Functional Constraints,” *PLoS One*, Vol. 3(6):e2246 (June 2008).**

Computer simulations of evolution such as Avida have been widely touted as having refuted intelligent design. But close scrutiny of these simulations reveal that they do not model true Darwinian processes because they are essentially pre-programmed to evolve complex systems. This peer-reviewed paper by ID-proponents attempts to present a computer simulation that fixes these defects by modeling Darwinian evolution in a biologically accurate manner, superior to that used by other evolutionary simulations such as Avida.

- **Michael Sherman, “Universal Genome in the Origin of Metazoa: Thoughts About Evolution,” *Cell Cycle*, Vol. 6(15):1873-1877 (August 1, 2007).**

This striking paper supports intelligent design advocates who view life as being “front-loaded” to allow for biological evolution. For example, the paper states, “This model has two major predictions, first that a significant fraction of genetic information in lower taxons must be functionally useless but becomes useful in higher taxons, and second that one should be able to turn on in lower taxons some of the complex latent developmental programs, e.g., a program of eye development or antibody synthesis in sea urchin.” In other words, lower taxa somehow have the genetic tools to produce systems that they do not have, but that do exist in higher taxa. As the article states: “Genes that are seemingly useless in sea urchin but are very useful in higher taxons exemplify excessive genetic information in lower taxons. It is unclear how such genetic complexity could have evolved.” When discussing the convergent use of pax-6 in widely diverse organisms, it states: “So, how does it happen that convergently evolved systems have the same developmental switches? These findings are very difficult to explain within the context of Darwinian ideas.” The author proposes a hypothesis where some pre-Cambrian ancestor that had “a Universal Genome that encodes all major developmental programs essential for every phylum of Metazoa emerged in a unicellular or a primitive multicellular organism.” This common ancestor then lost much genetic information in many lineages: “The proposed model of a Universal Genome implies that a lot of information encoded in genomes is not utilized in each individual taxon, and therefore is effectively useless.” The article suggests that microevolution is at work, but that Darwinian macroevolution cannot be credited with major innovations: “Furthermore, genetic evolution in combination with natural selection could define microevolution, however, within this model it is not responsible for the emergence of the major developmental programs.” This is an evolutionary model, but it challenges the sort of unguided and random evolution inherent to neo-Darwinism, and supports a front-loading intelligent design model.

- **Kirk K. Durston, David K. Y. Chiu, David L. Abel, Jack T. Trevors, “Measuring the functional sequence complexity of proteins,” *Theoretical Biology and Medical Modelling*, Vol. 4:47 (2007).**

This article devises a method of measuring the functional sequence complexity of proteins, which in turn permits “distinguishing between order, randomness, and biological function.” The authors suggest that “If genes can be thought of as information processing subroutines, then proteins can be analyzed in terms of the products of information interacting with laws of physics.” The metric of functional sequence complexity advanced by these authors is highly similar to the notion of complex and specified information.

- **Wolf-Ekkehard Lönnig and Heinz-Albert Becker, "Carnivorous Plants," in *Handbook of Plant Science*, Vol 2:1493-1498 (edited by Keith Roberts, John Wiley & Sons, 2007).**

This 2007 chapter on carnivorous plants by Lönnig and Becker in the John Wiley & Sons volume *Handbook of Plant Sciences* notes that “it appears to be hard even to imagine the clearcut selective advantages for all the thousands of postulated intermediate steps in a gradual scenario, not to mention the formulation and examination of scientific (i.e. testable) hypotheses for the origin of the complex carnivorous plant structures examined above.” They go on to favorably cite the work of Michael Behe, stating:

The reader is further invited to consider the following problem. Charles Darwin provided a sufficiency test for his theory (1859, p. 219): “If it could be demonstrated that any complex organ existed, which could not possibly have been formed by numerous, successive, slight modifications, my theory would absolutely break down.” Darwin, however, stated that he could “not find such a case.” Biochemist Michael J. Behe (1996, p. 39) has refined Darwin’s statement by introducing and defining his concept of “irreducible complexity”, specifying: “By *irreducibly complex* I mean a single system composed of several well-matched interacting parts that contribute to the basic function, wherein the removal of any one of the parts causes the system to effectively cease functioning.” Some biologists believe the trap mechanism(s) of *Utricularia* and several other carnivorous plant genera (*Dionaea*, *Aldrovanda*, *Genlisea*) come at least very near to “such a case” of irreducible complexity.

- **David L. Abel, “Complexity, self-organization, and emergence at the edge of chaos in life-origin models,” *Journal of the Washington Academy of Sciences*, Vol. 93:1-20 (2007).**

This article suggests that intelligent mind is responsible for the complexity of life, stating: “In computer science, only the programmer’s mind determines which way the switch knob is pushed. In evolution science we say that environmental selection ‘favors’ the fittest small groups. But selection is still the key factor, not chance and necessity. If physiodynamics set the switches, the switches would either be set randomly by heat agitation, or they would be set by force relationships and constants. Neither chance nor necessity, nor any combination of the two, can program. Chance produces only noise

and junk code. Law would set all of the switches the same way. Configurable switches must be set using 'choice with intent' if 'computational halting' is expected."

- **Felipe Houat de Brito, Artur Noura Teixeira, Otávio Noura Teixeira, Roberto C. L. Oliveira, "A Fuzzy Intelligent Controller for Genetic Algorithm Parameters," in *Advances in Natural Computation* (Licheng Jiao, Lipo Wang, Xinbo Gao, Jing Liu, Feng Wu, eds, Springer-Verlag, 2006); Felipe Houat de Brito, Artur Noura Teixeira, Otávio Noura Teixeira, Roberto C. L. Oliveira, "A Fuzzy Approach to Control Genetic Algorithm Parameters," *SADIO Electronic Journal of Informatics and Operations Research*, Vol. 7(1):12-23 (2007).**

This paper (published in two different venues) uses genetic algorithms that are controlled by an "intelligent agent based on fuzzy logic" and finds that such a method is more efficient than a random search typical of Darwinism. Citing the Intelligent Design and Evolution Awareness (IDEA) Center, it states: "The success achieved in the implementation of an intelligent agent controlling the evolutionary process is somewhat similar to the controversial approach of the Intelligent Design Theory, which is defended by many scientists as an answer to several aspects that are not well explained by the neo-Darwinist Theory."

- **Wolf-Ekkehard Lönnig, Kurt Stüber, Heinz Saedler, Jeong Hee Kim, "Biodiversity and Dollo's Law: To What Extent can the Phenotypic Differences between *Misopates orontium* and *Antirrhinum majus* be Bridged by Mutagenesis," *Bioremediation, Biodiversity and Bioavailability*, Vol. 1(1):1-30 (2007).**

This study attempts to trace the evolutionary history of two taxa of flowering plants that evolutionary biologists believe to be closely related. The authors tried to use mutagenesis experiments to cause the plants' traits to revert to a more "primitive" form, but found that such basic mutagenesis experiments were unable to cause the reversion of the taxa to the "'primitive' state." The authors have an explanation for their observations that explains a long-standing law of evolution, and supports the basic tenets of intelligent design: "since most new characters arise, not by simple additions but by integration of complex networks of gene functions rendering many systems to be irreducibly complex (Behe 1996, 2004; for a review, see Lönnig 2004), such systems cannot -- in agreement with Dollo's law -- simply revert to the original state without destroying the entire integration pattern guaranteeing the survival of a species." They conclude that, "for the rise of these taxa as well as for the inception of irreducible complex systems, the debate continues whether mutations and selection alone will be sufficient to produce all the new genetic functions and innovations necessary for the cytoplasm, membranes, and cell walls." The article favorably cites works from ID-friendly scientists such as Doug Axe's articles in *Journal of Molecular Biology*; Michael Behe's *Darwin's Black Box*; Behe and Snoke's 2004 article in *Protein Science*; David Berlinski's writings in *Commentary*; William Dembski's books *The Design Inference*, *No Free Lunch*, and *The Design Revolution*; Stephen C. Meyer's article in *Proceedings of the Biological Society of Washington*, and his work in *Darwinism, Design, and Public Education*; and also cites pro-ID entries from *Debating Design*.

- **Wolf-Ekkehard Lönnig, “Mutations: The Law of Recurrent Variation,” *Floriculture, Ornamental and Plant Biotechnology*, Vol. 1:601-607 (2006).**
Citing *Darwin’s Black Box* and other articles by Michael Behe about irreducible complexity, as well as the work of William Dembski and Stephen Meyer, this article states: “all the models and data recently advanced to solve the problem of completely new functional sequences and the origin of new organs and organ systems by random mutations have proved to be grossly insufficient in the eyes of many researchers upon close inspection and careful scientific examination.” Citing the work of Meyer, it further notes the “limits of the origin of species by mutations.”
- **David L. Abel and Jack T. Trevors, “Self-organization vs. self-ordering events in life-origin models,” *Physics of Life Reviews*, Vol. 3:211–228 (2006).**
This article, co-authored by a theoretical biologist and an environmental biologist, explicitly challenges the ability of Darwinian mechanisms or self-organizational models to account for the origin of the language-based chemical code underlying life. They explain that “evolutionary algorithms, neural nets, and cellular automata have not been shown to self-organize spontaneously into nontrivial functions.” The authors observe that life, “typically contains large quantities of prescriptive information.” They further argue that “[p]rescription requires choice contingency rather than chance contingency or necessity,” entailing a necessary appeal to an intelligent cause. Throughout the paper, the authors use positive arguments referencing the creative power of “agents” as they cite the work of Discovery Institute fellows and ID-theorists William Dembski, Charles Thaxton, and Walter Bradley. Critiquing models of self-organization, they conclude that “[t]he only self that can organize its own activities is a living cognitive agent.”
- **David L. Abel and Jack T. Trevors, “More than Metaphor: Genomes Are Objective Sign Systems,” *Journal of BioSemiotics*, Vol. 1(2):253-267 (2006).**
This article argues for intelligent design, observing that only intelligence capable of making choices can create the complexity we see in human beings. The authors state: “Neither chance contingency (quantified by Shannon theory) nor any yet-to-be-discovered law of nature can generate selection contingency. Yet selection contingency is abundantly evident throughout nature.” The sort of cause that is needed looks like this: “If the brain’s decision nodes were constrained by natural law, our decisions would not be real. If our choices were constrained by chance or necessity, we should stop holding engineers responsible for building collapses, and stop holding criminals responsible for their behavior. Real selection/choice contingency not only predates the existence of human metaphor and heuristic use of analogy, it produced human mentation.” According to the authors, “Sign systems in human experience arise only from choice contingency at successive decision nodes, not chance contingency or necessity.”

- **Øyvind Albert Voie, “Biological function and the genetic code are interdependent,” *Chaos, Solitons and Fractals*, Vol. 28:1000–1004 (2006).**

In this article, Norwegian scientist Øyvind Albert Voie examines an implication of Gödel’s incompleteness theorem for theories about the origin of life. Gödel’s first incompleteness theorem states that certain true statements within a formal system are unprovable from the axioms of the formal system. Voie then argues that the information processing system in the cell constitutes a kind of formal system because it “expresses both function and sign systems.” As such, by Gödel’s theorem it possesses many properties that are not deducible from the axioms which underlie the formal system, in this case, the laws of nature. He cites Michael Polanyi’s seminal essay “Life’s Irreducible Structure” in support of this claim. As Polanyi put it, “the structure of life is a set of boundary conditions that harness the laws of physics and chemistry their (the boundary conditions’) structure cannot be defined in terms of the laws that they harness.” As he further explained, “As the arrangement of a printed page is extraneous to the chemistry of the printed page, so is the base sequence in a DNA molecule extraneous to the chemical forces at work in the DNA molecule.” Like Polanyi, Voie argues that the information and function of DNA and the cellular replication machinery must originate from a source that transcends physics and chemistry. In particular, since as Voie argues, “chance and necessity cannot explain sign systems, meaning, purpose, and goals,” and since “mind possesses other properties that do not have these limitations,” it is “therefore very natural that many scientists believe that life is rather a subsystem of some Mind greater than humans.”

- **Kirk Durston and David K. Y. Chiu, “A Functional Entropy Model for Biological Sequences,” *Dynamics of Continuous, Discrete & Impulsive Systems: Series B Supplement* (2005).**

This peer-reviewed article by ID-proponents seeks to offer definitions of information that measure information in terms of functionality. The authors’ approach mirrors the concept of specified complexity. They explain that “The purpose of this paper is to show that Shannon entropy can also be redefined as a function of the joint patterns between data and functionality, thus incorporating a functional interpretation into the measure.” They explain that their methods can also be used to measure the degree of mutational changes necessary to convert one functional protein into another: “The difference in functional entropy between the two different sequences not only provides an estimate for the amount of information required to change the starting sequence into the final sequence, but it also calculates the estimated number of trials to achieve the final sequence in evolution” and thus “The functional entropy change calculated ... can be interpreted as a quantifier of evolutionary change.” Their paper experimentally tests their methods, calculating difference in functional entropy between a *Hox* enzyme found in insects and crustaceans, thought to be homologous. They write: “Since the novel function as expressed did not come into effect until all 6 mutations were in place, the evolutionary path was modeled as a random walk” and yielded a change of ~26 bits. According to Axe (2010), this of course pushes the limit of what can be produced by Darwinian evolution.

- **David L. Abel and Jack T. Trevors, “Three subsets of sequence complexity and their relevance to biopolymeric information,” *Theoretical Biology and Medical Modeling*, Vol. 2(29):1-15 (August 11, 2005).**

This article recognizes the important point that biological information must be defined in terms of the specific type of information it represents. Shannon information and Komologorov information are said to be inadequate measures of information. Instead, the authors recommend using functional sequence complexity, a concept essentially identical to specified complexity, to measure biological information. The article also refers to “choice contingency” entailing an “arbitrary intelligent choice” as a known cause: “Compression of language is possible because of repetitive use of letter and word combinations. Words correspond to reusable programming modules. The letter frequencies and syntax patterns of any language constrain a writer’s available choices from among sequence space. But these constraints are the sole product of arbitrary intelligent choice within the context of that language. Source and destination reach a consensus of communicative methodology before any message is sent or received. This methodology is called a language or an operating system. Abstract concept (‘choice contingency’) determines the language system, not ‘chance contingency,’ and not necessity (the ordered patterning of physical ‘laws.’)” It then argues that true organization, such as that studied in biology, requires this “choice contingency,” implying intelligent design: “Self-ordering phenomena are observed daily in accord with chaos theory. But under no known circumstances can self-ordering phenomena like hurricanes, sand piles, crystallization, or fractals produce algorithmic organization. Algorithmic ‘self-organization’ has never been observed despite numerous publications that have misused the term. Bone fide organization always arises from choice contingency, not chance contingency or necessity.”

- **John A. Davison, “A Prescribed Evolutionary Hypothesis,” *Rivista di Biologia/Biology Forum*, Vol. 98: 155-166 (2005).**

Otto Schindewolf once wrote that evolution postulates “a unique, historical course of events that took place in the past, is not repeatable experimentally and cannot be investigated in that way.” In this peer-reviewed article from an Italian biology journal, John A. Davison agrees with Schindewolf. Since “[o]ne can hardly expect to demonstrate a mechanism that simply does not and did not exist,” Davison attempts to find new explanations for the origin of convergence among biological forms. Davison contends that “[t]he so-called phenomenon of convergent evolution may not be that at all, but simply the expression of the same preformed ‘blueprints’ by unrelated organisms.” While discussing many remarkable examples of “convergent evolution,” particularly the marsupial and placental saber-toothed cats, Davison is unmistakable in his meaning. The evidence, he writes, “bears, not only on the questions raised here, but also, on the whole issue of Intelligent Design.” Davison clearly implies that this evidence is expected under an intelligent design model, but not under a Neo-Darwinian one.

- **Douglas D. Axe, “Estimating the Prevalence of Protein Sequences Adopting Functional Enzyme Folds,” *Journal of Molecular Biology*, Vol. 341:1295–1315 (2004).**

This experimental study shows that functional protein folds are extremely rare, finding that “roughly one in 10^{64} signature-consistent sequences forms a working domain” and that the “overall prevalence of sequences performing a specific function by any domain-sized fold may be as low as 1 in 10^{77} .” Axe concludes that “functional folds require highly extraordinary sequences.” Since Darwinian evolution only preserves biological structures that confer a functional advantage, it would be very difficult for such a blind mechanism to produce functional protein folds. This research also shows that there are high levels of specified complexity in enzymes, a hallmark indicator of intelligent design. Axe himself has confirmed that this study adds to the evidence for intelligent design: “In the 2004 paper I reported experimental data used to put a number on the rarity of sequences expected to form working enzymes. The reported figure is less than one in a trillion trillion trillion trillion trillion. Again, yes, this finding does seem to call into question the adequacy of chance, and that certainly adds to the case for intelligent design.” See [Scientist Says His Peer-Reviewed Research in the Journal of Molecular Biology “Adds to the Case for Intelligent Design”](#).

- **Michael Behe and David W. Snoke, “Simulating evolution by gene duplication of protein features that require multiple amino acid residues,” *Protein Science*, Vol. 13 (2004).**

In this article, Lehigh University biochemist Michael Behe and University of Pittsburgh physicist Snoke show how difficult it is for unguided evolutionary processes to take existing protein structures and add novel proteins whose interface compatibility is such that they could combine functionally with the original proteins. According to their analysis, mechanisms in addition to standard Darwinian processes are required to generate many protein-protein interactions:

The fact that very large population sizes— 10^9 or greater—are required to build even a minimal MR feature requiring two nucleotide alterations within 10^8 generations by the processes described in our model, and that enormous population sizes are required for more complex features or shorter times, seems to indicate that the mechanism of gene duplication and point mutation alone would be ineffective, at least for multicellular diploid species, because few multicellular species reach the required population sizes. Thus, mechanisms in addition to gene duplication and point mutation may be necessary to explain the development of MR features in multicellular organisms.

By demonstrating inherent limitations to unguided evolutionary processes, this work gives indirect scientific support to intelligent design and bolsters Behe’s case for intelligent design in answer to some of his critics.

- **Wolf-Ekkehard Lönnig, “Dynamic genomes, morphological stasis, and the origin of irreducible complexity,” in Valerio Parisi, Valeria De Fonzo, and Filippo Aluffi-Pentini eds., *Dynamical Genetics* (2004).**

Biology exhibits numerous invariants -- aspects of the biological world that do not change over time. These include basic genetic processes that have persisted unchanged for more than three-and-a-half billion years and molecular mechanisms of animal ontogenesis that have been constant for more than one billion years. Such invariants, however, are difficult to square with dynamic genomes in light of conventional evolutionary theory. Indeed, Ernst Mayr regarded this as one of the great unsolved problems of biology. In this paper Dr. Wolf-Ekkehard Lönnig, Senior Scientist in the Department of Molecular Plant Genetics at the Max-Planck-Institute for Plant Breeding Research (now retired), employs the design-theoretic concepts of irreducible complexity (as developed by Michael Behe) and specified complexity (as developed by William Dembski) to elucidate these invariants, accounting for them in terms of an explicit intelligent design hypothesis.

- **Stephen C. Meyer, “The origin of biological information and the higher taxonomic categories,” *Proceedings of the Biological Society of Washington*, Vol. 117(2):213-239 (2004) ([HTML](#)).**

This article argues for intelligent design as an explanation for the origin of the Cambrian fauna. Not surprisingly, it created an international firestorm within the scientific community when it was published. (See David Klinghoffer, “[The Branding of a Heretic](#),” *Wall Street Journal*, Jan. 28, 2005, as well as the following website by the editor who oversaw the article’s peer-review process: <http://www.richardsternberg.net/>.) The treatment of the editor who sent Meyer’s article out for peer-review is a striking illustration of the sociological obstacles that proponents of intelligent design encounter in publishing articles that explicitly defend the theory of intelligent design.

John Angus Campbell and Stephen C. Meyer, *Darwinism, Design, and Public Education* (East Lansing, Michigan: Michigan State University Press, 2003; published as part of the peer-reviewed Rhetoric and Public Affairs Series).

This is a collection of interdisciplinary essays that addresses the scientific, philosophical, and educational controversies concerning the theory of intelligent design. According to Michigan State University Press’s original description of the book: “Darwinism, Design, and Public Education examines ID as a science, a philosophy, and a movement for educational reform. In this book, leading design theorists present their scientific case for intelligent design, their criticisms of contemporary Darwinism and their arguments for a pluralistic controversy-based model of science education.” The book was peer-reviewed by a philosopher of science, a rhetorician of science, and a professor in the biological sciences from an Ivy League university. The book includes five scientific articles advancing the case for the theory of intelligent design, the contents of which are summarized below.

- **S. C. Meyer, “Dna and the Origin of Life: Information, Specification and Explanation,” pp. 223-285, in John Angus Campbell and Stephen C. Meyer, *Darwinism, Design, and Public Education* (East Lansing, Michigan: Michigan State University Press, 2003.**
 Meyer contends that intelligent design provides a better explanation than competing chemical evolutionary models for the origin of the information present in large bio-macromolecules such as DNA, RNA, and proteins. Meyer shows that the term information as applied to DNA connotes not only improbability or complexity but also specificity of function. He then argues that neither chance nor necessity, nor the combination of the two, can explain the origin of information starting from purely physical-chemical antecedents. Instead, he argues that our knowledge of the causal powers of both natural entities and intelligent agency suggests intelligent design as the best explanation for the origin of the information necessary to build a cell in the first place.
- **M. J. Behe, “Design in the Details: The Origin of Biomolecular Machines,” pp. 287-302, in John Angus Campbell and Stephen C. Meyer, *Darwinism, Design, and Public Education* (East Lansing, Michigan: Michigan State University Press, 2003.**
 Behe sets forth a central concept of the contemporary design argument, the notion of “irreducible complexity.” Behe bases his argument on a consideration of phenomena studied in his field, biochemistry, including systems and mechanisms that display complex, interdependent, and coordinated functions. Such intricacy, Behe argues, defies the causal power of natural selection acting on random variation, the “no end in view” mechanism of neo-Darwinism. On the other hand, he notes that irreducible complexity is a feature of systems that are known to be designed by intelligent agents. He thus concludes that, compared to Darwinian theory, intelligent design provides a better explanation for the presence of irreducible complexity in the molecular machines of the cell.
- **P. Nelson and J. Wells, “Homology in Biology: Problem for Naturalistic Science and Prospect for Intelligent Design,” pp. 303-322, in John Angus Campbell and Stephen C. Meyer, *Darwinism, Design, and Public Education* (East Lansing, Michigan: Michigan State University Press, 2003.**
 Paul Nelson and Jonathan Wells reexamine the phenomenon of homology, the structural identity of parts in distinct species such as the pentadactyl plan of the human hand, the wing of a bird, and the flipper of a seal, on which Darwin was willing to rest his entire argument. Nelson and Wells contend that natural selection explains some of the facts of homology but leaves important anomalies (including many so-called molecular sequence homologies) unexplained. They argue that intelligent design explains the origin of homology better than do mechanisms cited by advocates of neo-Darwinism.
- **S. C. Meyer, M. Ross, P. Nelson, P. Chien, “The Cambrian Explosion: Biology’s Big Bang,” pp. 323-402, , in John Angus Campbell and Stephen C. Meyer, *Darwinism, Design, and Public Education* (East Lansing, Michigan: Michigan State University Press, 2003.**

Meyer, Ross, Nelson, and Chien show that the pattern of fossil appearance in the Cambrian period contradicts the predictions or empirical expectations of neo-Darwinian (and punctuationalist) evolutionary theory. They argue that the fossil record displays several features -- a hierarchical top-down pattern of appearance, the morphological isolation of disparate body plans, and a discontinuous increase in information content -- that are strongly reminiscent of the pattern of evidence found in the history of human technology. Thus, they conclude that intelligent design provides a better, more causally adequate explanation of the origin of the novel animal forms present in the Cambrian explosion.

- **W. A. Dembski, "Reinstating Design Within Science," pp. 403-418, , in John Angus Campbell and Stephen C. Meyer, *Darwinism, Design, and Public Education* (East Lansing, Michigan: Michigan State University Press, 2003).**
Dembski argues that advances in the information sciences have provided a theoretical basis for detecting the prior action of an intelligent agent. Starting from the commonsense observation that we make design inferences all the time, Dembski shows that we do so on the basis of clear criteria. He then shows how those criteria, complexity and specification, reliably indicate intelligent causation. He gives a rational reconstruction of a method by which rational agents decide between competing types of explanation, those based on chance, physical-chemical necessity, or intelligent design. Since he asserts we can detect design by reference to objective criteria, Dembski also argues for the scientific legitimacy of inferences to intelligent design.
- **Frank J. Tipler, "Intelligent Life in Cosmology," *International Journal of Astrobiology*, Vol. 2(2): 141-148 (2003).**
This paper by Tulane mathematician and cosmologist Frank Tipler observes that teleological explanations are live possibilities within physics. Tipler also contends that the universe is set up to permit the existence of life, and that the universe seems guided by an ultimate goal inherent it. The implication, as Tipler writes, is that the evolution of life has been guided by that goal, rather than being entirely random.
- **David L. Abel, "Is Life reducible to complexity?," *Fundamentals of Life*, Chapter 1.2 (2002).**
This article suggests that explaining the functional complexity in life requires a force that can make choices: "Progress in understanding the derivation of bioinformation through natural processes will come only through elucidating more detailed mechanisms of selection pressure 'choices' in biofunctional decision-node sequences. The latter is the subject of both 'BioFunction theory' and the more interdisciplinary 'instruction theory'. ... Life, then, is not only not reducible to complexity; it is not even reducible to FSC! Life is a symphony of dynamic, highly integrated, algorithmic processes yielding homeostatic metabolism, development, growth, and reproduction (ignoring the misgivings of those few life-origin theorists with mule fixations!). But as Yockey argues, it remains to be seen whether such highly sophisticated algorithmic processes can exist apart from the linear, segregatable, digital, FSC instructions observed at the helm of all known empirical

life.” The author argues that “The key to life-origin research lies in uncovering the mechanisms whereby these productive algorithmic programming choices were made and recorded in nucleic acid.” He compares the processes that generated life to those that generate computer programming: “Selection is exactly what is found in computer algorithms. Correct choices at each successive decision node alone produce sophisticated software. RSC strings are pragmatically distinguished from FSC strings by virtue of the fact that RSC strings are almost never observed to do anything useful in any context. FSC strings, on the other hand, can be counted on to contribute specific utility.”

- **David K.Y. Chiu and Thomas W.H. Lui, “Integrated Use of Multiple Interdependent Patterns for Biomolecular Sequence Analysis,” *International Journal of Fuzzy Systems*, Vol. 4(3):766-775 (September 2002).**
Citing the work of William Dembski, the opening paragraph of this article reads: “Detection of complex specified information is introduced to infer unknown underlying causes for observed patterns. By complex information, it refers to information obtained from observed pattern or patterns that are highly improbable by random chance alone. We evaluate here the complex pattern corresponding to multiple observations of statistical interdependency such that they all deviate significantly from the prior or null hypothesis. Such multiple interdependent patterns when consistently observed can be a powerful indication of common underlying causes. That is, detection of significant multiple interdependent patterns in a consistent way can lead to the discovery of possible new or hidden knowledge.”
- **Michael J. Denton, Craig J. Marshall, and Michael Legge, “The Protein Folds as Platonic Forms: New Support for the pre-Darwinian Conception of Evolution by Natural Law,” *Journal of Theoretical Biology*, Vol. 219: 325-342 (2002).**
These researchers reach a conclusion that is thoroughly teleological and non-Darwinian. The authors look to laws of form embedded in nature as possessing the power to guide the formation of biological structures. The intelligent design research program reflected here is broad yet certainly recognizable, positing design as a feature programmed into nature.
- **Wolf-Ekkehard Lönnig and Heinz Saedler, “Chromosome Rearrangement and Transposable Elements,” *Annual Review of Genetics*, Vol. 36:389–410 (2002).**
This article examines the role of transposons in the abrupt origin of new species and the possibility of a partly predetermined generation of biodiversity and new species. The authors’ approach is non-Darwinian, and they cite favorably the work of design theorists Michael Behe and William Dembski, acknowledging that some biological systems are irreducibly complex.
- **Douglas D. Axe, “Extreme Functional Sensitivity to Conservative Amino Acid Changes on Enzyme Exteriors,” *Journal of Molecular Biology*, Vol. 301:585-595 (2000).**
This study published by molecular biologist Douglas Axe, now at the Biologic Institute, challenges the widespread idea that high species-to-species variation in the amino-acid

sequence of an enzyme implies modest functional constraints. Darwinists commonly assume that such variation indicates low selection pressure at the variable amino acid sites, allowing many mutations with little effect. Axe's research shows that even when mutations are restricted to these sites, they are severely disruptive, implying that proteins are highly specified even at variable sites. According to this work, sequences diverge not because substantial regions are free from functional constraints, but because selection filters most mutations, leaving only the harmless minority. By showing functional constraints to be the rule rather than the exception, it raises the question of whether chance can ever produce sequences that meet these constraints in the first place. Axe himself has confirmed that this study adds to the evidence for intelligent design: "I concluded in the 2000 JMB paper that enzymatic catalysis entails 'severe sequence constraints.' The more severe these constraints are, the less likely it is that they can be met by chance. So, yes, that finding is very relevant to the question of the adequacy of chance, which is very relevant to the case for design." See [Scientist Says His Peer-Reviewed Research in the Journal of Molecular Biology "Adds to the Case for Intelligent Design"](#).

- **Solomon Victor and Vijaya M. Nayak, "Evolutionary anticipation of the human heart," *Annals of the Royal College of Surgeons of England*, Vol. 82:297-302 (2000).**
This article argues that intelligent design is recognizable in the human heart, stating: "Comparative anatomy points to a design and a Designer. Surgeons, anatomists and anyone studying the human form and function have an unsurpassed opportunity to ponder over the wonders of creation and contemplate the basic questions: where did we come from? why are we here? and where are we going?"
- **Solomon Victor, Vijaya M. Nayek, and Raveen Rajasingh, "Evolution of the Ventricles," *Texas Heart Institute Journal*, Vol. 26:168-175 (1999).**
This article concludes that "there is a design in the evolution of the venous connections of the heart, pectinate muscles, atrioventricular valves, left ventricular tendons, outflow tracts, and great arteries." But the version of "evolution" it presents is decidedly non-Darwinian, as it notes that evolution appears to be goal-directed by a designer: "One neglected aspect in the study of evolution is that of anticipation. Fish atria and ventricles appear to have a built-in provision for becoming updated to the human 4-chambered structure. This transformation is achieved in stages: the truncus yields the great arteries, appropriate shifting takes place in the great arteries, the left ventricle decreases in sponginess and increases in the size of its lumen, the chordopapillary apparatus becomes more sophisticated, the coronary circulation undergoes changes, and the ventricular septal defect closes." The article closes by stating, "This evolutionary progression points to a master design and plan for countless millennia."
- **William A. Dembski, *The Design Inference: Eliminating Chance through Small Probabilities* (Cambridge: Cambridge University Press, 1998).**
This book was published by Cambridge University Press and peer-reviewed as part of a distinguished monograph series, Cambridge Studies in Probability, Induction, and

Decision Theory. The editorial board of the series includes members of the National Academy of Sciences as well as a Nobel laureate, John Harsanyi, who shared the prize in 1994 with John Nash, protagonist of the film *A Beautiful Mind*. Commenting on the ideas in *The Design Inference*, well-known physicist and science writer Paul Davies remarked: “Dembski’s attempt to quantify design, or provide mathematical criteria for design, is extremely useful. I’m concerned that the suspicion of a hidden agenda is going to prevent that sort of work from receiving the recognition it deserves.” Quoted in Larry Witham, *By Design* (San Francisco: Encounter Books, 2003), p. 149.

- **R. Kunze, H. Saedler, and W.-E. Lönnig, “Plant Transposable Elements,” in *Advances in Botanical Research*, Vol. 27:331-470 (Academic Press, 1997).**
This peer-reviewed chapter from an academic book on plant research favorably references Michel Behe’s concept of irreducible complexity. After noting that “some major problems have to be solved for gene duplications to be of fundamental evolutionary significance,” it cites to Behe’s 1996 book *Darwin’s Black Box* to justify the question: “What could be the selective advantage of the intermediate (‘still unfinished’) reaction chains?” The authors further state that “examples of ‘irreducibly complex systems’” are found in biology.
- **Michael Behe, *Darwin’s Black Box: The Biochemical Challenge to Evolution* (New York: The Free Press, 1996).**
In this book Behe develops a critique of the mechanism of natural selection and a positive case for the theory of intelligent design based upon the presence of “irreducibly complex molecular machines” and circuits inside cells. Though this book was published by The Free Press, a trade press, the publisher subjected the book to standard scientific peer-review by several prominent biochemists and biological scientists.
- **Charles B. Thaxton, Walter L. Bradley, Roger L. Olsen, *The Mystery of Life’s Origin: Reassessing Current Theories* (New York: Philosophical Library, 1984; Dallas, Texas: Lewis & Stanley Publishing, 4th ed., 1992).**
In this book Thaxton, Bradley and Olsen develop a seminal critique of origin of life studies and develop a case for the theory of intelligent design based upon the information content and “low-configurational entropy” of living systems.
- **Stanley L. Jaki, “Teaching of Transcendence in Physics,” *American Journal of Physics*, Vol. 55(10):884-888 (October 1987).**
This article from the *American Journal of Physics* seeks to help educators understand how they can teach students about the evidence for transcendence in the universe. The article assumes that a transcendent realm exists beyond the universe and that the universe can plausibly be said to reflect design.

- **Granville Sewell, “Postscript,” in *Analysis of a Finite Element Method: PDE/PROTRAN* (New York: Springer Verlag, 1985).**

In this article appearing in a 1985 technical reference book, mathematician Granville Sewell compares the complexity found in the genetic code of life to that of a computer program. He recognizes that the fundamental problem for evolution is the “problem of novelties” which in turn raises the question “How can natural selection cause new organs to arise and guide their development through the initial stages during which they present no selective advantage?” Sewell explains how a typical Darwinist will try to bridge both functional and fossil gaps between biological structures through “a long chain of tiny improvements in his imagination,” but the author notes that “the analogy with software puts his ideas into perspective.” Major changes to a species require the intelligent foresight of a programmer. Natural selection, a process that is “unable to plan beyond the next tiny mutation,” could never produce the complexity of life.

- **William G. Pollard, “Rumors of transcendence in physics,” *American Journal of Physics*, Vol. 52 (10) (October 1984).**

In this peer-reviewed paper, nuclear physicist William G. Pollard notes that Big Bang cosmology requires some kind of transcendent reality. Pollard argues that the scientific justification for this transcendent domain can be found in quantum mechanics because universal laws and constants are finely-tuned to permit the existence of advanced life, which point to an intelligent source, a mind, as designing the universe.

Category 2: Peer-Edited or Editor-Reviewed Articles Supportive of Intelligent Design Published in Scientific Journals, Scientific Anthologies and Conference Proceedings

Selected Publications from this Category

- Jonathan Wells, “Not Junk After All: Non-Protein-Coding DNA Carries Extensive Biological Information,” pp. 210-231, in Robert J. Marks II, Michael J. Behe, William A. Dembski, Bruce L. Gordon, and John C. Sanford eds., *Biological Information: New Perspectives* (Singapore: World Scientific, 2013).
- Michael J. Behe, “Getting There First: An Evolutionary Rate Advantage for Adaptive Loss-of-Function Mutations,” pp. 450-473, in Robert J. Marks II, Michael J. Behe, William A. Dembski, Bruce L. Gordon, and John C. Sanford eds., *Biological Information: New Perspectives* (Singapore: World Scientific, 2013).
- Jonathan Wells, “Do Centrioles Generate a Polar Ejection Force?” *Rivista di Biologia / Biology Forum*, Vol. 98:71-96 (2005).
- Heinz-Albert Becker and Wolf-Ekkehard Lönnig, “Transposons: Eukaryotic,” *Encyclopedia of Life Sciences* (John Wiley & Sons, 2005).
- Scott A. Minnich and Stephen C. Meyer, “Genetic analysis of coordinate flagellar and type III regulatory circuits in pathogenic bacteria,” *Proceedings of the Second International Conference on Design & Nature*, Rhodes, Greece, edited by M.W. Collins and C.A. Brebbia (Ashurst, Southampton, United Kingdom: WIT Press, 2004).
- Michael Behe, “Irreducible Complexity: Obstacle to Darwinian Evolution,” pp. 352-370, in William A. Dembski and Michael Ruse, eds., *Debating Design: From Darwin to DNA* (Cambridge, United Kingdom: Cambridge University Press, 2004).
- Douglas D. Axe and Ann K. Gauger, “Explaining Metabolic Innovation: Neo-Darwinism versus Design,” pp. 489-507, in Robert J. Marks II, Michael J. Behe, William A. Dembski, Bruce L. Gordon, and John C Sanford eds., *Biological Information: New Perspectives* (Singapore: World Scientific, 2013).
- Granville Sewell, “A Mathematician’s View of Evolution,” *The Mathematical Intelligencer*, Vol. 22(4) (2000).

Annotated Bibliography of Publications in this Category

- **William A. Dembski, Winston Ewert, and Robert J. Marks II, “A General Theory of Information Cost Incurred by Successful Search,” pp. 26- 63, in Robert J. Marks II, Michael J. Behe, William A. Dembski, Bruce L. Gordon, and John C. Sanford eds., *Biological Information: New Perspectives* (Singapore: World Scientific, 2013).**

This paper provides a general explanation of a methodology for measuring “active information,” or the amount of information that is added to a random search to aid in finding a search target. This foundational paper is theoretical, aiming to establish mathematically that without active information being added, a search can perform, on average, no better than a random search:

When a search with probability q of success displaces a baseline search with probability p of success where $q > p$, conservation of information states that raising the probability of successful search by a factor of $q / p (>1)$ incurs an information cost of at least $\log(q / p)$. Conservation of information shows that information, like money, obeys strict accounting principles.

In other words, a “natural” search -- one that operates blindly -- cannot, on average, find the target any faster than a random search working without active information. To increase the probability of finding the target, “active information” must be added. This “conservation” of information operates in a law-like fashion that would prevent unguided mechanisms, like Darwinian processes, from improving the search. This is the essence of Dembski’s “No Free Lunch” theorem. As Marks explains in the introduction to the volume, a search can be simplified “only by access to some source of information.” That source, of course, requires intelligence. This methodology can then be applied to real-world situations, as it allows them to measure the extent to which computerized simulations of evolution are generating new information, or simply using information introduced by the programmer, not generated by the program.

- **Winston Ewert, William A. Dembski, and Robert J. Marks II, “Tierra: The Character of Adaptation,” pp. 105-138, in Robert J. Marks II, Michael J. Behe, William A. Dembski, Bruce L. Gordon, and John C. Sanford eds., *Biological Information: New Perspectives* (Singapore: World Scientific, 2013).**

In this paper, Ewert, Dembski, and Marks apply the sort of methodology developed in their first paper (“A General Theory of Information Cost Incurred by Successful Search”). This study investigates Tierra, one of the earliest computerized simulations of evolution, developed by Thomas Ray in 1989. According to Ray, Tierra is capable of modeling the evolution of complexity, as supposedly occurred in events like the Cambrian explosion. As Ewert, Dembski, and Marks explain, in Ray’s mind “once evolution (whether biological or artificial) has produced a Cambrian explosion, the rest of evolution should proceed easily.” They observe, however, that after 20+ years of people using Tierra, the widely agreed conclusion “is that Tierra did not produce a Cambrian explosion or open-ended evolution.” They observe that “Tierran evolution can be characterized as an initial

period of high activity producing a number of novel adaptations followed by barren stasis,” and thus ask why Tierra stopped producing new features. They explain:

A closer look at Tierran evolution reveals an important characteristic of the adaptations. Tierra started with a designed ancestor to seed the population. In other words, it presupposed something like an origin of life and was concerned with the development of complexity after that point. The ancestor provides initial information to Tierra. Adaptations primarily consist of rearranging or removing that information. Open-ended evolution requires adaptations which increase information. However, such adaptations are rare in Tierra. Tierra’s informational trajectory is reversed from what evolution requires. It is dominated by loss and rearrangement with only minimal new information instead of being dominated by the production of new information with minimal cases of removal or rearrangement of information. Long term evolutionary progress is dependent on the generation of new information.

Does Tierra actually produce new information? Ewert, Dembski, and Marks studied the workings of Tierra in detail and found:

In a majority of the cases we see that evolution proceeded by deleting instructions. There are some new instructions inserted, but these are much smaller than the changes in other areas. As a result, we can clearly see that Tierran evolution is dominated by information-reducing mutations. ... The interesting behaviors produced by Tierra are created mostly by rearranging the information seeded into the simulation by its designer.

Thus, they found that Tierra was in a sense front-loaded -- or intelligently designed -- to stably evolve:

Tierra also derives some information from the environment in which it runs. Ray was concerned about the brittleness of machine code, and accordingly made specific design decisions. Additionally, the original instruction set was created by choosing exactly the instructions which were used in the ancestor. This results in the Tierra instruction set being specifically tuned to the problem it faces. This work has not attempted to investigate the implications of these decisions, but it is our opinion that the Tierran evolution is substantially assisted through them.

Nonetheless, they observe, “The author of Tierra sought to create a digital Cambrian explosion whereby the power of the evolutionary process was unleashed. It is agreed that Tierra did not succeed in accomplishing this feat. Rather, the evolutionary activity within Tierra dies after only a transitory period. No Cambrian explosion occurs.”

- **George Montañez, Robert J. Marks II, Jorge Fernandez, and John C. Sanford, “Multiple Overlapping Genetic Codes Profoundly Reduce the Probability of Beneficial Mutation,”**

pp. 139-167, in Robert J. Marks II, Michael J. Behe, William A. Dembski, Bruce L. Gordon, and John C. Sanford eds., *Biological Information: New Perspectives* (Singapore: World Scientific, 2013).

How rare are beneficial mutations? This paper attempts to address that crucial question, since its answer “determines both the speed and the direction of genetic change.” The authors point out that “If beneficial mutations are extremely rare, this profoundly limits the *rate and range* of all forward genetic change.” In addressing the rarity of beneficial mutations, they observe that “DNA sequences are typically ‘poly-functional’,” meaning that any given nucleotide “can contribute to multiple overlapping codes simultaneously.” They observe that “overlapping protein-coding sequences” are now considered “typical,” meaning that it’s common for a single base pair to “affect multiple traits simultaneously and interactively.” To address the rarity of beneficial mutations, they perform computer modeling on functional DNA sequences that contain multiple overlapping codes, and describe the likelihoods of a given mutation being beneficial towards at least one code, or having a net-benefit, meaning it “is a mutation that improves more codes than it disrupts.” They conclude that poly-functional codes will rarely incur universally beneficial mutations:

[W]ithin all poly-functional nucleotide sites, essentially all “beneficial mutations” will at best be ambiguously beneficial, being beneficial at just one level, but simultaneously being deleterious at one or more levels. Therefore at any poly-functional nucleotide site, a “beneficial” mutation will almost always still consistently have deleterious effects, systematically eroding the total amount of information in the entire information system.

They conclude that “that increasing either the number of overlapping codes or the degree of optimization has negative effects on the probability of producing a beneficial mutation.” Indeed, they argue that “The growing evidence for polyfunctional DNA therefore suggests that unambiguously beneficial mutations should be vanishingly rare,” and “it is difficult to understand how poly-functional DNA could arise through random isolated mutations.”

- **Granville Sewell, “Entropy, Evolution and Open Systems,” pp. 168-178, in Robert J. Marks II, Michael J. Behe, William A. Dembski, Bruce L. Gordon, and John C. Sanford eds., *Biological Information: New Perspectives* (Singapore: World Scientific, 2013).** In this article, Granville Sewell address the argument from defenders of Darwinian evolution that the second law of thermodynamics poses no problems for their model. They argue if entropy increases elsewhere in the universe, then it can decrease in open systems such as the Earth. Sewell calls this the “compensation” argument, and explains why it is seriously flawed:

It is widely argued that the spectacular local decreases in entropy that occurred on Earth as a result of the origin and evolution of life and the development of human intelligence are not inconsistent with the second law of thermodynamics, because

the Earth is an open system and entropy can decrease in an open system, provided the decrease is compensated by entropy increases outside the system. I refer to this as the compensation argument, and I argue that it is without logical merit, amounting to little more than an attempt to avoid the extraordinary probabilistic difficulties posed by the assertion that life has originated and evolved by spontaneous processes. To claim that what has happened on Earth does not violate the fundamental natural principle behind the second law, one must instead make a more direct and difficult argument.

Sewell elaborates that “the whole idea of compensation, whether by distant or nearby events, makes no sense logically: an extremely improbable event is not rendered less improbable simply by the occurrence of ‘compensating’ events elsewhere. According to this reasoning, the second law does not prevent scrap metal from reorganizing itself into a computer in one room, as long as two computers in the next room are rusting into scrap metal -- and the door is open. (Or the thermal entropy in the next room is increasing, though I am not sure how fast it has to increase to compensate for computer construction!)” He concludes: “The ‘compensation’ counter-argument was produced by people who generalized the model equation for isolated systems, but forgot to generalize the equation for non-isolated systems.” His generalized model would be as follows: “If an increase in order is extremely improbable when a system is closed, it is still extremely improbable when the system is open, unless something is entering which makes it not extremely improbable.”

Sewell’s argument is not that the second law is necessarily a barrier to Darwinian evolution since, “Of course, one can still argue that the spectacular increase in order seen on Earth is consistent with the underlying principle behind the second law because what has happened here is not really extremely improbable...But one would think that at least this would be considered an open question, and those who argue that it really is extremely improbable, and thus contrary to the basic principle underlying the second law of thermodynamics, would be given a measure of respect, and taken seriously by their colleagues.”

- **Andy C. McIntosh, “Information and Thermodynamics in Living Systems,” pp. 179-201, in Robert J. Marks II, Michael J. Behe, William A. Dembski, Bruce L. Gordon, and John C. Sanford eds., *Biological Information: New Perspectives* (Singapore: World Scientific, 2013).**

This paper seeks to understand the origin of information in life, and proposes “an entirely different paradigm whereby the non-material message is accepted as being of an origin outside the area of physical investigation, but that its effect can readily be seen in the organisation of the molecular machinery in living organisms.” In McIntosh’s view, “Rather than the material and energy forming the information system as advocated by evolutionary philosophy, the non-material informational message expressed in the coded ordering of nucleotides is actually the mechanism of constraining the material itself.” In his view, “Understanding the thermodynamics of this machinery shows that it

is thermodynamically impossible both to form such machinery (abiogenesis) without intelligence, and that the laws of thermodynamics prohibit any formation of new machinery which is not there already or latently coded for in the DNA (evolutionary development).”

- **Jonathan Wells, “Not Junk After All: Non-Protein-Coding DNA Carries Extensive Biological Information,” pp. 210-231, in Robert J. Marks II, Michael J. Behe, William A. Dembski, Bruce L. Gordon, and John C. Sanford eds., *Biological Information: New Perspectives* (Singapore: World Scientific, 2013).**

Citing numerous examples of functionality for non-coding DNA, in this paper Jonathan Wells argues that “the notion of ‘junk DNA’ is obsolete, and the amount of biological information in the genome far exceeds the information in protein-coding regions.” Wells uncovers various lines of evidence in support of this claim.

First, there are the conclusions of the ENCODE project which suggest that there is “widespread transcription of non-protein-coding DNA.” According to Wells, this “suggests probable function; so does sequence conservation.”

This evidence, however, is somewhat circumstantial. Thus Wells observes as a second point that “[t]here is also direct evidence for specific functions of non-protein-coding RNAs.” He gives many examples of how non-coding RNA performs specific functions in cells, including:

- Regulating gene expression.
- Alternative splicing, allowing the construction of many new transcripts. As Wells explains, “Alternative splicing plays an essential role in the differentiation of cells and tissues at the proper times during embryo development, and many alternatively spliced RNAs occur in a developmental-stage-and tissue-specific manner.”
- Introns not only regulate gene expression, but “also encode many of the small RNAs essential for the processing of ribosomal RNAs, as well as the regulatory elements associated with such RNA-coding sequences.”
- “Non-protein-coding RNAs are essential for chromatin organization, and non-protein-coding RNAs have been shown to affect gene expression by modifying chromatin structure.”
- “Pseudogenes are transcribed into non-protein-coding RNAs that in some cases regulate the expression of the corresponding protein-coding genes.”

Wells also observes that the nucleotide sequence of DNA is not the only way that non-coding DNA can specify functions:

The genome functions hierarchically, and the order of nucleotides in protein-coding and non-protein-coding DNA constitutes only the first level of that hierarchy. The length of DNA sequences (even non-protein-coding ones) is a second level; chromatin organization is a third level; and the position of chromosomes within the nucleus is a

fourth. There is evidence that DNA functions at the second, third, and fourth levels in ways that are independent of the precise nucleotide sequence.

He concludes that even as we find more and more functionality for non-coding DNA, other non-DNA-based sources of information are being discovered in living cells.

- **Donald Johnson, “Biocybernetics and Biosemiosis,” pp. 402-413, in Robert J. Marks II, Michael J. Behe, William A. Dembski, Bruce L. Gordon, and John C. Sanford eds., *Biological Information: New Perspectives* (Singapore: World Scientific, 2013).**
Can biology be studied through computer science? In this paper, computer scientist and chemist Donald Johnson argues that we can. He writes:

Any serious origin-of-life or origin-of-species scenario must explain the origin of the required biological information. It is argued that each protein arises as the result of the execution of a genuine computer program. The creation of a functional protein via the mutation/selection paradigm lacks support from information science. Those who understand the reality of bioinformation, especially the prescriptive information of biocybernetics, will be able to incorporate that understanding into new models that will lead to a more complete understanding of life.

Johnson recognizes that “The vital nature of information in life has been downplayed by most materialists, since functional information has no feasible cause from physicality (though infeasible scenarios have been speculated).” However, “biology is an information science since all of the defining characteristics of biology are controlled by life’s information processing systems.” But life doesn’t just contain information, for “life uses common operating systems, programming languages, and devices.” Indeed, Johnson argues “[t]here are many components of life that can thus be classified as computers or components of computers,” such as the transcription/translation system.

Johnson also observes that life contains semiotic systems, which “is a system made of two independent worlds that are connected by the conventional rules of a code. ... made of three distinct entities: signs, meanings and code.” Again the transcription/translation system provides a prime example, but “[s]ince information is non-material, there have been no feasible scenarios for production of semiotic systems from physicality.”

- **Jed C. Macosko and Amanda M. Smelser, “An Ode to the Code: Evidence for Fine-Tuning in the Standard Codon Table,” pp. 418-434, in Robert J. Marks II, Michael J. Behe, William A. Dembski, Bruce L. Gordon, and John C. Sanford eds., *Biological Information: New Perspectives* (Singapore: World Scientific, 2013).**
It’s well-established that the standard codon table (SCT) of the genetic code is finely-tuned in a way “that minimizes harmful effects of mutations and mistranslations while maximizing the encoding of multiple messages into a single sequence.” But how did this

optimization arise? This paper argues that “external intelligence better explains the origin of the SCT.”

According to the authors, Francis Crick’s “frozen accident” model, fails because “the SCT could have ended up with any arbitrary structure,” and thus they cannot explain the optimization of the code. Other models seek to explain the optimization through material mechanisms, such as chemical interactions “between amino acids and their respective trinucleotide codons.” These attempts have failed since “the preference for codon versus anticodon involvement appears random.” The authors also observe that materialistic explanations of the genetic code “do not explain the origin of the machinery that is responsible for converting mRNA information into amino acid sequences” and thus “theories for the origin of the coding machinery are abundant and are generally viewed as extremely speculative.”

But just how finely-tuned is the genetic code? They propose that the SCT is optimized in multiple ways, which are both “optimal and are orthogonal, i.e. the optimality of one would not necessarily lead to the optimality of the others.” These include:

1) similar amino acids are coded by similar codons thus minimizing the impact of errors, 2) the family/non-family symmetry minimizes mistranslations while maximizing tRNA usage efficiency, 3) the stop codons are related to commonly occurring amino acids in a way that optimizes second-layer codes, and 4) methionine is an optimal initiating amino acid due to its minimized energy for exiting the ribosome.

Given this degree of fine-tuning, they calculate that if the SCT is the “best of all possible codes,” then natural selection can only work if there were a population of competing codes approaching 10^{84} . This, they observe, is “a ludicrous population size, considering that 10^{84} carbon atoms are a trillion, trillion, trillion times more massive than the earth.” They conclude intelligent design is the best explanation since “the general pattern of intelligence producing finely-tuned, optimized effects is well-known and well-studied,” and that an ID-based paradigm could have anticipated and accelerated the discovery of fine-tuning of the genetic code.

- **Michael J. Behe, “Getting There First: An Evolutionary Rate Advantage for Adaptive Loss-of-Function Mutations,” pp. 450-473, in Robert J. Marks II, Michael J. Behe, William A. Dembski, Bruce L. Gordon, and John C. Sanford eds., *Biological Information: New Perspectives* (Singapore: World Scientific, 2013).**

This paper elaborates on some of Behe’s arguments from his 2010 paper in *Quarterly Review of Biology*, in which he reviewed molecular mechanisms involved in adaptations in microorganisms documented in the literature. He found there that such adaptations almost always involved loss or diminishment of function. In this paper Behe explores the implications of these observations for population genetics, and finds they pose a major challenge to Darwinian evolution.

Behe begins by observing that at the molecular level, far more mutations will cause loss-of function (LOF) than will cause a gain-of-function (GOF):

It is very often possible to eliminate a molecular function by a variety of mutations. GOF mutations, on the other hand, are generally much more specific, sometimes being produced in only one way.

For example, Behe explores potential molecular mechanisms that can confer resistance to malaria in humans. One molecular mechanism involves creating a new binding site -- which Behe calls a GOF mutation. This requires very specific mutations. But other mechanisms work because they prevent production of a functional protein. Behe observes that there are many mutations that prevent the gene from functioning. This example helps explain why LOF mutations are far more common than GOF mutations. He concludes: "Because of the many ways in which a gene can be altered to lose function, the LOF mutation would have a rate several orders of magnitude greater than that of the GOF mutation for the duplicated gene." What are the implications for neo-Darwinian evolution?

If different types of mutations (say, GOF mutations or LOF mutations) can confer some particular advantage on an organism, the LOF mutation is likely to become fixed before the GOF mutation since "LOF mutations always possess a rate advantage over GOF mutations if the respective selection coefficients are equal." After reviewing various molecular adaptations observed in experiments reported in the literature, Behe argues: "Both experimental laboratory work over the past few decades and recent genomic studies of adaptation in natural populations attest to the importance, even dominance, of LOF mutations in short term evolutionary episodes." His work helps make sense of this situation:

The work presented in this paper helps show why this should be the case. Functional genetic elements such as genes and regulatory regions are built of multiple nucleotides, and a substantial fraction of mutations to these elements will cause them to lose their function. Thus the LOF mutation rate can be orders of magnitude greater than the nucleotide substitution rate. On the other hand, GOF mutations tend to be quite specific. So the rate for adaptive GOF mutations tends to be equal or very similar to the nucleotide mutation rate. As shown here, for some population size regions and for some values for the ratio of selection coefficients, the greater rate of mutation to the adaptive state for LOF versus GOF gives adaptive LOF mutations an intrinsic edge over adaptive GOF mutations.

This suggests that when Darwinian evolution is at work, it tends to diminish or destroy molecular functions rather than creating them. Behe closes with a quote from two biologists who observe that "there clearly are complex structures that are gained during evolution ... and we currently know little about how this process takes place." The

implication, of course, is that a process like Darwinian evolution, which tends to break or diminish functional molecular elements, is not a viable explanation for how these complex structures arose in the first place.

- **Jonathan Wells, “The Membrane Code: A Carrier of Essential Biological Information That Is Not Specified by DNA and Is Inherited Apart from It,” pp. 474-488, in Robert J. Marks II, Michael J. Behe, William A. Dembski, Bruce L. Gordon, and John C. Sanford eds., *Biological Information: New Perspectives* (Singapore: World Scientific, 2013).** In this paper, Jonathan Wells argues that “a genetic program is not sufficient for embryogenesis: biological information outside of DNA is needed to specify the body plan of the embryo and much of its subsequent development.” Wells elaborates:

Some of that information is in cell membrane patterns, which contain a two-dimensional code mediated by proteins and carbohydrates. These molecules specify targets for morphogenetic determinants in the cytoplasm, generate endogenous electric fields that provide spatial coordinates for embryo development, regulate intracellular signaling, and participate in cell-cell interactions. Although the individual membrane molecules are at least partly specified by DNA sequences, their two-dimensional patterns are not. Furthermore, membrane patterns can be inherited independently of the DNA.

Does this epigenetic information pose a problem for neo-Darwinism? Wells thinks it does:

One could speculate that accidental changes in membrane patterns -- analogous to accidental mutations in DNA -- could provide the missing raw materials for evolution. Yet two- and three-dimensional information-carrying patterns are likely to entail more specified complexity than the one-dimensional information in DNA sequences, making beneficial “mutations” in such patterns much less probable than beneficial mutations in DNA. At the very least, calculations of the time required for evolution will now have to take into account these higher dimensions of biological information.

Thus, any viable model for the origin of biological information must explain not just the information in gene-coding DNA, but also the origin of the information in non-coding DNA, and the origin of epigenetic information. That, as even some evolutionary biologists are starting to acknowledge, is a tall order.

- **Douglas D. Axe and Ann K. Gauger, “Explaining Metabolic Innovation: Neo-Darwinism versus Design,” pp. 489-507, in Robert J. Marks II, Michael J. Behe, William A. Dembski, Bruce L. Gordon, and John C Sanford eds., *Biological Information: New Perspectives* (Singapore: World Scientific, 2013).**

In this paper, Axe and Gauger review the results of their own prior research, and the research of others, and examine six obstacles to Darwinian explanations of metabolic

complexity. They suggest that a design-based paradigm offers the best solution to those obstacles.

In discussing the first obstacle, the authors cite experimental work showing that duplicate genes are much more likely to be silenced, “Because gene expression is costly, it cannot be assumed that weakly converted enzyme functions isolated by laboratory selection would provide net selective benefit in wild populations.” Intelligent design could provide a better explanation because such innovations require a goal-directed cause that looks beyond immediate fitness costs that go along with preserving a non-advantageous duplicate gene.

The second and third obstacles pertain to the fact that “billions of years might be necessary” for features to generate arise that require “rare mutations or rare combinations of mutations” in order to function. They review their own research which suggests that multiple mutations would be required for even modest protein-to-protein conversions to occur. They conclude: “enzymatic innovations requiring more than two specific mutations in a spare gene (provided by a duplication event) are implausible in neo-Darwinian terms.” Some goal directed process is required that can generate these complex adaptations.

The third problem found that producing even one new protein could strain the probabilistic resources of the Darwinian mechanism. But to produce metabolic complexity would require “multiple enzymatic innovations.” In their view, “This poses a severe challenge for neo-Darwinism. Mechanisms that have been proposed in attempts to meet this challenge, such as retrograde evolution, or serial duplication and recruitment do not match the actual distribution of protein domains across and within pathways.” Again, a goal-directed process seems necessary to solve this problem since “Useful innovations tend to require the simultaneous solution of multiple new problems, which means they tend to be compound innovations.” Like the fifth problem, they note that many simultaneous mutations would be required to produce new protein folds – leading to a similar obstacle, and solution.

The sixth problem is a novel one. Some proteins are necessary for their own production, leading to what Axe and Gauger call “causal circularity.” This presents something of a chicken-and-egg problem for Darwinism:

[I]n order to conceive of an evolutionary origin of biotin biosynthesis, we must suppose that prior to this origin either A) cells were making their membranes without biotin, or B) cells had an abiotic source of biotin.

The authors realize that life itself presents such an obstacle: “Since life is a prerequisite for all biosynthesis, any biosynthetic product that is necessary for life in its present form is also necessary for its own biosynthesis in modern life. So causal circularity exists for all essential biosynthetic products.” Yet again, only a complex process, capable of working

in a top-down fashion to coordinate multiple parts, can solve this problem. As a goal-directed process, intelligent design stands apart from unguided Darwinian evolution, and can uniquely provide the kind of innovative solutions necessary for complex life.

- **A. C. McIntosh, “Functional Information and Entropy in Living Systems,” *Design and Nature III: Comparing Design in Nature with Science and Engineering*, Vol. 87 (Ashurt, Southampton, United Kingdom: WIT Transactions on Ecology and the Environment, WIT Press, 2006).**

This paper explores the proper way to measure information and entropy in living organisms. Citing the work of Stephen Meyer, the author argues that random mutations cannot increase order in a living system: “[R]andom mutations always have the effect of increasing the disorder (or what we will shortly define as logical entropy) of any particular system, and consequently decreasing the information content. What is evident is that the initial information content rather than being small must in fact be large, and is in fact vital for any process to work to begin with. The issue of functional complexity and information is considered exhaustively by Meyer who argues that the neo-Darwinist model cannot explain all the appearances of design in biology.” McIntosh continues, explaining that only teleology -- intelligent design -- can explain the increases in information that generate observed biological complexity: “Even within the neo-Darwinist camp the evidence of convergence (similarity) in the suggested evolutionary development of disparate phylogeny has caused some writers to consider ‘channelling’ of evolution. Such thinking is a tacit admission of a teleological influence. That information does not increase by random changes (contrary to Dawkins’ assertion) is evident when we consider in the following section, the logical entropy of a biochemical system.” He concludes that goal-directed processes, or teleonomy, are required: “There has to be previously written information or order (often termed teleonomy’) for passive, non-living chemicals to respond and become active.”

- **Jonathan Wells, “Do Centrioles Generate a Polar Ejection Force?” *Rivista di Biologia / Biology Forum*, Vol. 98:71-96 (2005).**

Molecular biologist Jonathan Wells writes in the Italian biology journal *Rivista di Biologia* that the cell may be viewed and studied as a designed system with engineered machines. Showing the heuristic value of intelligent design, he writes: “Instead of viewing centrioles through the spectacles of molecular reductionism and neo-Darwinism, this hypothesis assumes that they are holistically designed to be turbines. ... What if centrioles really are tiny turbines? This is much easier to conceive if we adopt a holistic rather than reductionistic approach, and if we regard centrioles as designed structures rather than accidental by-products of neo-Darwinian evolution. If centrioles really are turbines, then fluid exiting through the blades would cause them to rotate clockwise when viewed from their proximal ends.” Wells hypothesizes that such approaches may lead to understandings of the workings of centrioles, perhaps even uncovering some causes of cancer.

- **Heinz-Albert Becker and Wolf-Ekkehard Lönnig, “Transposons: Eukaryotic,”** *Encyclopedia of Life Sciences* (John Wiley & Sons, 2005).

This encyclopedia entry recounts that some biological systems may be irreducibly complex, stating: “A general difficulty to be mentioned in this context (but not inherent in the selfish DNA hypothesis) is that mutation and selection may not be the full explanation for the origin of species; i.e. the factors of the neo-Darwinian scenario may find their limits, for example, in the generation of ‘irreducibly complex structures’ (Behe, 1996). This is a term used to describe structures that, according to Behe and co-workers, cannot be explained by a piecemeal production via intermediate steps.” The article elaborates on Behe’s argument stating, “Among the examples discussed by Behe are the origins of (1) the cilium, (2) the bacterial flagellum with filament, hook and motor embedded in the membranes and cell wall and (3) the biochemistry of blood clotting in humans.” The article then proposes that additional systems may challenge Darwinian explanations, stating: “Moreover, the traps of *Utricularia* (and some other carnivorous plant genera) as well as several further apparatuses in the animal and plant world appear to pose similar problems for the modern synthesis (joints, echo location, deceptive flowers, etc.). Up to now, none of these systems has been satisfactorily explained by neo-Darwinism. Whether accelerated TE activities with all the above named mutagenic consequences can solve the questions posed remains doubtful.”
- **Scott A. Minnich and Stephen C. Meyer, “Genetic analysis of coordinate flagellar and type III regulatory circuits in pathogenic bacteria,”** *Proceedings of the Second International Conference on Design & Nature*, Rhodes, Greece, edited by M.W. Collins and C.A. Brebbia (Ashurst, Southampton, United Kingdom: WIT Press, 2004).

This article underwent conference peer review to be included in this peer-edited volume of proceedings. Minnich and Meyer do three important things in the paper. First, they refute a popular objection to Michael Behe’s argument for the irreducible complexity of the bacterial flagellum. Second, they suggest that the Type III Secretory System present in some bacteria, rather than being an evolutionary intermediate to the bacterial flagellum, probably represents a degenerate form of the same. Finally, they argue explicitly that compared to the neo-Darwinian mechanism, intelligent design better explains the origin of the bacterial flagellum. As the authors explain, “In all irreducibly complex systems in which the cause of the system is known by experience or observation, intelligent design or engineering played a role in the origin of the system.”
- **William A. Dembski, “The Logical Underpinnings of Intelligent Design,”** pp. 311-330, in **William A. Dembski and Michael Ruse, eds.,** *Debating Design: From Darwin to DNA* (Cambridge, United Kingdom: Cambridge University Press, 2004).

In this article, Dembski outlines his method of design detection. He proposes a rigorous way of identifying the effects of intelligent causation and distinguishing them from the effects of undirected natural causes and material mechanisms. Dembski shows how the presence of specified complexity or “complex specified information” provides a reliable marker of prior intelligent activity. He also responds to a common criticism made

against his method of design detection, namely that design inferences constitute “an argument from ignorance.”

- **Walter L. Bradley, “Information, Entropy, and the Origin of Life,” pp. 331-351, in William A. Dembski and Michael Ruse, eds., *Debating Design: From Darwin to DNA* (Cambridge, United Kingdom: Cambridge University Press, 2004).**

Walter Bradley is a mechanical engineer and polymer scientist. In the mid 1980s he co-authored what supporters consider a seminal critique of origin of life studies in the book *The Mystery of Life's Origins*. Bradley and his co-authors also developed a case for the theory of intelligent design based upon the information content and “low-configurational entropy” of living systems. In this chapter he updates that work. He clarifies the distinction between configurational and thermal entropy, and shows why materialistic theories of chemical evolution have not explained the configurational entropy present in living systems, a feature that Bradley takes to be strong evidence of intelligent design.

- **Michael Behe, “Irreducible Complexity: Obstacle to Darwinian Evolution,” pp. 352-370, in William A. Dembski and Michael Ruse, eds., *Debating Design: From Darwin to DNA* (Cambridge, United Kingdom: Cambridge University Press, 2004).**

In this essay Behe briefly explains the concept of irreducible complexity and reviews why he thinks it poses a severe problem for the Darwinian mechanism of natural selection. In addition, he responds to several criticisms of his argument for intelligent design from irreducible complexity and several misconceptions about how the theory of intelligent design applies in biochemistry. In particular he discusses several putative counterexamples that some scientists have advanced against his claim that irreducibly complex biochemical systems demonstrate intelligent design. Behe turns the table on his critics, arguing that such examples actually underscore the barrier that irreducible complexity poses to Darwinian explanations, and, if anything, show the need for intelligent design.

- **Stephen C. Meyer, “The Cambrian Information Explosion: Evidence for Intelligent Design,” pp. 371-391, in William A. Dembski and Michael Ruse, eds., *Debating Design: From Darwin to DNA* (Cambridge, United Kingdom: Cambridge University Press, 2004).**

Meyer argues for design on the basis of the Cambrian explosion, the geologically sudden appearance of new animal body plans during the Cambrian period. Meyer notes that this episode in the history of life represents a dramatic and discontinuous increase in the complex specified information of the biological world. He argues that neither the Darwinian mechanism of natural selection acting on random mutations nor alternative self-organizational mechanisms are sufficient to produce such an increase in information in the time allowed by the fossil evidence. Instead, he suggests that such increases in specified complex information are invariably associated with conscious and rational activity, that is, with intelligent design.

- **Granville Sewell, “A Mathematician’s View of Evolution,” *The Mathematical Intelligencer*, Vol. 22(4) (2000) ([HTML](#)).**

Mathematician Granville Sewell explains that Michael Behe’s arguments against neo-Darwinism from irreducible complexity are supported by mathematics and the quantitative sciences, especially when applied to the problem of the origin of new genetic information. Sewell notes that there are “a good many mathematicians, physicists and computer scientists who...are appalled that Darwin’s explanation for the development of life is so widely accepted in the life sciences.” Sewell compares the genetic code of life to a computer program -- a comparison also made by computer gurus such as Bill Gates and evolutionary biologists such as Richard Dawkins. He notes that experience teaches that software depends on many separate functionally coordinated elements. For this reason “[m]ajor improvements to a computer program often require the addition or modification of hundreds of interdependent lines, no one of which makes any sense, or results in any improvement, when added by itself.” Since individual changes to part of a genetic program typically confer no functional advantage (in isolation from many other necessary changes to other portions of the genetic code), Sewell argues that improvements to a genetic program require the intelligent foresight of a programmer. Undirected mutation and selection will not suffice to produce the necessary information.

Category 3: Articles Supportive of Intelligent Design Published in Peer-Reviewed Philosophy Journals, or Peer-Reviewed Philosophy Books Supportive of Intelligent Design

Selected Publications from this Category

- Michael Behe, “Reply to my Critic: A Response to Reviews of *Darwin’s Black Box: The Biochemical Challenge to Evolution*,” *Biology and Philosophy*, Vol. 16, 685–709, (2001).
- Michael Behe, “Self-Organization and Irreducibly Complex Systems: A Reply to Shanks and Joplin,” *Philosophy of Biology*, Vol. 67(1):155-162 (March, 2000).
- William Lane Craig, “Barrow and Tipler on the Anthropic Principle vs. Divine Design,” *British Journal for the Philosophy of Science*, Vol. 38: 389-395 (1988).

Partially Annotated Bibliography of Publications in this Category

- **Michael C. Rea, *World without Design: The Ontological Consequences of Naturalism* (Oxford University Press, 2004).**
- **William Lane Craig, “Design and the Anthropic Fine-Tuning of the Universe,” in *God and Design: The Teleological Argument and Modern Science*, pp. 155-177. (Neil Manson ed., London: Routledge, 2003).**
- **Michael Behe, “Reply to my Critic: A Response to Reviews of *Darwin’s Black Box: The Biochemical Challenge to Evolution*,” *Biology and Philosophy*, Vol. 16, 685–709, (2001).** In this article published in the mainstream journal *Biology and Philosophy*, Michael Behe defends his views supporting intelligent design as stated in *Darwin’s Black Box*.
- **Del Ratzsch, *Nature, Design, and Science: The Status of Design in Natural Science* (State University of New York Press, 2001).**
- **William Lane Craig, “The Anthropic Principle,” in *The History of Science and Religion in the Western Tradition: An Encyclopedia*, pp. 366-368 (Gary B. Ferngren, general ed., Garland Publishing, 2000).**
- **Michael Behe, “Self-Organization and Irreducibly Complex Systems: A Reply to Shanks and Joplin,” *Philosophy of Biology*, Vol. 67(1):155-162 (March, 2000).** Michael Behe defends his arguments for irreducible complexity against the criticisms of various Darwinian scientists.
- **William Lane Craig, “Barrow and Tipler on the Anthropic Principle vs. Divine Design,” *British Journal for the Philosophy of Science*, Vol. 38: 389-395 (1988).**

- William Lane Craig, "God, Creation, and Mr. Davies," *British Journal for the Philosophy of Science*, Vol. 37: 168-175 (1986).