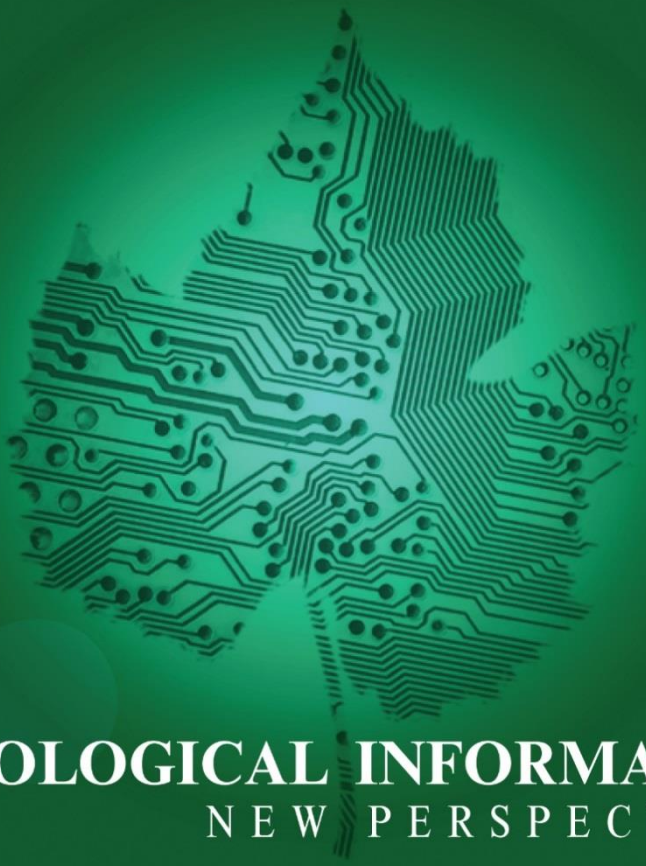


BIOLOGICAL INFORMATION
NEW PERSPECTIVES

A Synopsis and Limited Commentary

Dr. J. C. Sanford

Biological Information – New Perspectives

A Synopsis and Limited Commentary

J.C. Sanford
Courtesy Associate Professor
Department of Horticulture
Cornell University

In 2011 a symposium was held at Cornell University, entitled Biological Information - New Perspectives. The proceedings of that symposium (by the same title), have now been published by World Scientific. That volume was edited by Drs. Marks, Behe, Dembski, Gordon, and Sanford.

Dr. Sanford, organizer of the symposium and a co-editor of the proceedings, wrote this booklet to make the information within the proceedings more generally accessible. This booklet greatly condenses the information within the 563 pages of the much larger volume, and uses much less technical language. In addition, this booklet contains limited commentary on the significance of each of the 24 scientific papers. Lastly, this booklet includes credentials and a short bio for the first author of each chapter.

The author of this booklet thanks all 29 scientists who contributed to the proceedings, including his fellow co-editors, for their important contributions to this milestone work. Apologies are given in advance for possible shortcomings in adequately distilling and commenting on each individual paper.

Published by FMS Publications
Waterloo, NY, USA

Introduction

In the summer of 2013, the book *Biological Information – New Perspectives* was published (http://www.worldscientific.com/worldscibooks/10.1142/8818). The book includes the proceedings of a symposium of the same name, which was held at Cornell University in the spring of 2011. What is the significance of this symposium and its proceedings? More importantly, what is the significance of biological information itself?

The proceedings include the research findings of 29 scientists who represent a diverse spectrum of scientific disciplines, including information theory, computer science, numerical simulation, thermodynamics, evolutionary theory, whole organism biology, developmental biology, molecular biology, genetics, physics, biophysics, mathematics, and linguistics. These scientists generally agreed on three crucial points:

1. Information is the key to understanding life. Within the simplest cell there exists an immense flow of information through a mind-boggling system of information networks. There is constant and multidirectional communication between proteins, RNAs, and DNAs, and these biological information networks are in many ways comparable to the internet.

2. These biological information systems appear to greatly surpass human information technologies. Such information systems cannot possibly operate until all the countless components of the system are in place - including hardware, software, multiple languages, storage/transmission of communicable prescriptive information units, error testing/correction systems, designated senders/receivers, etc. Such systems must be comprehensive and coherently integrated before they can effectively operate.

3. The enormous amounts of information found within any cell, and the irreducibly complex nature of information systems, can no longer rationally be attributed to just the mutation/selection process. New perspectives are needed that might help us better understand the nature, origin, and maintenance of biological information.

Biological Information – New Perspectives brings into serious question the long held neo-Darwinian paradigm, which has claimed for over a century that mutation/selection can explain all aspects of the biological realm. In light of the new evidence presented

in *Biological Information – New Perspectives*, it is necessary that biologists begin to re-examine neo-Darwinian theory.

A major limitation of this book is that its papers are so technical that most readers will not be able to readily absorb them. The book contains 24 high-level, rigorous, and usually exhaustive scientific research papers, written by experts in a wide range of scientific disciplines. For this reason each of the book's chapters (papers) tends to be too long and too technical for most non-specialists to absorb. Indeed, the average college professor may have considerable trouble understanding many of these papers which fall outside of his/her area of specialization. In order to make the information in the book more generally accessible, I have written my own view of the highlights of the book and its general significance. This synopsis attempts to boil down and briefly summarize each paper's highlights – using less technical language. I hope this synopsis will help non-specialists appreciate the significance of these authors' exciting new findings.

Because the authors contributing to this work were requested to avoid any lengthy philosophical discourse, the broader implications of the authors' findings were usually left un-spoken. For this reason, I attempt to capture the take-home message of each paper. In some instances I will doubtless miss the mark, and in these cases I apologize in advance. Additionally, in this synopsis I take the liberty of adding a limited amount of personal commentary.

Putting the Pieces Together

Summarizing *Biological Information – New Perspectives* is challenging because it contains 24 highly technical scientific papers which contain a huge amount of scholarly material covering a wide range of topics. However, I believe there are three general themes which can be used to tie together the papers in these proceedings. These themes are: **1) the amazing extent and sophistication of biological information; 2) the many difficulties associated with creating such biological information solely using the mutation/selection process; 3) the extreme difficulties associated with preventing the systematic degradation of such biological information, given only the mutation/selection process.** Therefore, I have organized this summary along the lines of these themes, rather than following the sequence of the actual symposium sessions.

Theme 1: The Nature of Biological Information

Nine of the papers included within these proceedings primarily investigated the *nature of biological information*. These papers, taken collectively, show us that within any living cell there is a vast amount of biological information, and more importantly - a huge array of *information systems*. The labyrinth of information networks within any cell greatly surpasses what scientists could have imagined a decade ago. We are experiencing an explosion in our awareness of what biological information actually entails. It entails many types of information, encoded by many languages, manifested at many different biological levels. We are talking about layer upon layer of information. The information networks that enable life are extraordinarily complex, diverse, dynamic, and multi-dimensional. These biological information networks are comparable in many ways to today's internet. However, while the internet reflects a vast tangle of disjointed websites, a cell's enormous array of information networks involves an astounding degree of integration and remarkable unity of purpose (to enable life). We have only scratched the surface in terms of understanding all the aspects of biological information, but it is already clear that biological information systems greatly surpass the best human information technologies. All scientists should be in awe of what is now emerging. All of this biological information demands an explanation in terms of its origin and preservation.

Below are short thumbnail sketches of these first nine papers, and a brief comment on each paper's significance. Each summary is prefaced by a link to the complete on-line chapter.

Biological Information – What is It?

Werner Gitt, Robert Crompton and Jorge Fernandez

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0001

Dr. Gitt (et al.), a world-recognized specialist in information theory, provides an overview of *functional information*. Dr. Gitt et al. show that biological information is exactly the same type of information that we use every day in our electronic communications. Biological information is what makes life alive, in the same way information gives life to our computers, the internet, and modern society. Like any type of real-world information, biological information entails language (symbolic representation and grammar), meaning (an informative message or specification), and purpose (an expected result). Dr. Gitt et al. show that information is itself a *non-material entity* - it is neither matter nor energy. Mere matter cannot create information or information systems. So how did biological information arise?

Significance: *It is irrational to believe that inanimate matter, without any guiding force, can spontaneously give rise to complex information systems embodying language, meaning, and purpose. It is our universal experience that these things arise only through the operation of intelligence. If information/language/meaning/purpose* ***do not*** *imply intelligence, then what do we mean by the word "intelligence"?*

Pragmatic Information

John W. Oller, Jr.

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0003

Dr. Oller, an expert on language acquisition, measurement of language proficiency, and the diagnosis of language disorders and related issues, shows that biological information systems, like the information that can be expressed in natural languages (English, Chinese, etc.), depends on a deeply layered hierarchy of inter-connected sign systems. The signs at the highest rank are richest in content. For instance, in natural languages we discover that sounds and syllables get their distinctive values from the meanings they help to distinguish in words, phrases, sentences, and higher linguistic units of structure. The key to Dr. Oller's argument is the simple mathematical fact that the number of possible strings at any given level in any natural language, or any language-like biological signaling system, grows exponentially as we progress up the hierarchy of information layers.

This is a profound insight. Every step up to a higher hierarchical level creates an explosion of possible strings (there are 26 English letters, but hundreds of thousands of English words, and innumerable possible English sentences). But with each step upward, the ratio of valid (meaningful) information strings versus meaningless nonsense strings plummets, quickly approaching zero. Therefore in order to create (or discern) meaning at higher and higher levels of language requires more and more intelligence. As we move up the language hierarchy, the requirement for the operation of intelligence does not increase linearly - it increases exponentially.

Significance: *Language is arguably the strongest single evidence for the presence of intelligence. The existence of many types of very high-level languages imbedded throughout all biological systems strongly points to an underlying intelligence.*

An Ode to the Code: Evidence for Fine-Tuning in the Standard Codon Table

Jed C. Macosko and Amanda M. Smelser

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0018

Dr. Macosko, an expert in biophysics, along with co-author Smelser, shows that the genetic code (the most basic language in the cell), is an incredibly optimized code. The 64 possible DNA triplets (codons), symbolically code instructions for protein synthesis

(the 64 codons encode 20 amino acids plus start and stop messages). This coding system is essentially universal for all living things. Historically, it was thought this universal code was arbitrary – merely a "frozen accident". It is now clear the code is not arbitrary; it is extremely optimized – the best possible code from among millions of possibilities.

Significance: *The evidence presented by Macosko and Smelser strongly argues that the genetic code had to have been already established and optimized BEFORE the first living cell could have come into being.*

Not Junk After All: Non-Protein-Coding DNA Carries Extensive Biological Information

Jonathan Wells

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0009

Molecular biologist Jonathan Wells reviews the scientific literature on non-protein-coding DNA, which makes up about 98% of the human genome. Some have called this "junk DNA" and argued that it is simply useless debris that has accumulated in the course of evolution. Some Darwinists have argued that junk DNA provides evidence for Darwinian theory and is evidence against intelligent design (since an intelligent designer would presumably not have littered organisms' DNA with so much useless debris). Wells shows, however, that much non-protein-coding DNA has been demonstrated to serve various biological functions, and that the list of demonstrated functions is growing. Wells has subsequently been vindicated by results from the ENCODE (for "Encyclopedia of DNA Elements") Project, which concluded in 2012 that upwards of 80% of human DNA is biologically functional. It is probable that the remaining 20% of the genome is functional. The collapse of the long-standing doctrine that higher genomes are primarily "junk DNA" represents a major paradigm shift.

Significance: *The amount of biological information that requires explanation is exploding. The term "junk DNA" has been used for decades as a dismissive term, meant to trivialize biological information, but it is now clear that our DNA, including the non-protein-coding parts of it, is an incredibly sophisticated information network.*

The Membrane Code: A Carrier of Essential Biological Information That is Not Specified by DNA and Is Inherited Apart from It

Jonathan Wells

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0021

Jonathan Wells (as above) reviews the evidence that cell membranes carry biological information that is necessary for embryo development but is not specified by DNA sequences. Membrane information is embodied in patterns that provide targets for the localization of intracellular molecules, global spatial coordinates in the form of electric

fields, and an extracellular "sugar code" that mediates interactions with other cells. Membrane patterns constitute a whole new layer of biological information, which cannot be reduced to DNA sequences or explained by DNA mutations, and thus cannot be understood in neo-Darwinian terms.

Significance: *The existence of whole new types of biological information (which transcend classic DNA-based genetic systems) greatly amplifies the explanatory deficiencies of neo-Darwinian theory. In addition to the membrane code, we have the splicing code, the methylation code, the histone code, the epigenetic code, etc. Neo-Darwinian theory cannot explain these newly understood information systems. How did they arise? How are they coordinated?*

A New Model of Intracellular Communication Based on Coherent, High Frequency Vibrations in Molecules

L. Dent

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0019

Dr. Dent, an expert in electrophysiology, explores the possibility of an entirely new level of biological networking within cells based upon molecular vibrational frequencies. This is the first cellular model which suggests that molecules may interact at a distance. This line of thinking raises the prospect of letting us go beyond the standard "billiard ball" model of random collisions of molecules within the cell. It appears quite clear that there has to be more going on within cells than simple Brownian motion. Otherwise many reactions would be much too slow (i.e., DNA replication). This is because so many molecules must sequentially "fly in" to just the right spot at just the right moment at just the right angle - then dock, react, and "fly away" to their next designated destination, making room for the next incoming molecule.

Significance: *The existence of multiple new categories of biological information, including this possible new vibrational communication system between molecules at a distance, is extremely exciting. Such systems could never be explained by mutation/selection, because like epigenetic systems and the membrane code, they must transcend DNA-based genetics.*

Multiple Overlapping Genetic Codes Profoundly Reduce the Probability of Beneficial Mutation

George Montañez, Robert J. Marks II, Jorge Fernandez and John C. Sanford

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0006

Montañez (et al.), a PhD candidate in the field of Machine Learning, shows that there are multiple overlapping codes (messages) within the genome. These authors show that diverse codes are extensively overlapping within the DNA sequence. This means that a

typical nucleotide can simultaneously contribute to multiple messages and multiple types of information systems. Therefore, most nucleotides are *poly-functional* – and so most nucleotides must be *poly-constrained*. It is demonstrated mathematically that this amazing reality profoundly limits the frequency of potential mutations which are truly beneficial. To the extent there is significant overlap in genetic codes, almost every mutation that is beneficial for one code will be deleterious for one or more other overlapping codes. Unambiguously beneficial mutations (not deleterious in any code) must therefore be extremely rare. To make matters worse, the vast majority of mutations which are truly beneficial will *not be subject to effective selection*. This is because of the selection threshold problem, wherein all very low-impact mutations become un-selectable (http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0011). Any truly beneficial mutations that might arise will fail to be selectively amplified, except where they simultaneously have a biological benefit above the selection threshold. Since almost all beneficial mutations make only a miniscule contribution to total fitness, truly beneficial mutations which are actually selectable must be vanishingly rare.

Significance: *Overlapping codes represent a type of data compression that computer scientists can only dream of. How could overlapping codes have ever arisen? Once in place, how could they ever be improved? Unambiguously beneficial mutations which are actually subject to selection must be vanishingly rare. How then do complex biological specifications arise?*

Biocybernetics and Biosemiosis

Donald Johnson

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0017

Dr. Johnson, having PhDs in both Computer Science and Biology, demonstrates that the information networks found within living cells are remarkably similar to computer networks. He shows that along with DNA, every protein and RNA simultaneously constitutes both hardware and software. As living algorithms, such information molecules simultaneously encode their own basic sequence, folding, transport, and biological function.

Significance: *Nobody thinks that computer networks (including the associated hardware, software, language, and specified meaning), could ever arise spontaneously. So is it reasonable to think that vastly superior biological information systems, occurring just above the atomic level, could arise by any type of Darwinian trial/error process?*

DNA.EXE: A Sequence Comparison between Human Genome and Computer Code

Josiah Seaman

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0016

Seaman (a computer scientist and a candidate for a PhD in bioinformatics), has expertise in data visualization, and shows that the architecture of higher genomes is remarkably similar to the architecture of executable computer code. In both information systems, strikingly similar repeating elements are seen (especially tandem repeat sequences). This strongly suggests that repeat elements found within higher genomes are not "junk DNA". He likens the genome of the cell to the *hard drive* of the cell, and describes the RNA and protein of the cell as the cell's *RAM*.

Significance: *Tandem repeats within genomes have historically been used as evidence for "junk DNA" and are cited as proof that the genome came together via a haphazard process. But nearly identical tandem repeats are also found throughout executable computer code. The tandem repeats in computer code are certainly not junk - they contain essential information. The tandem repeats in executable computer code never arise haphazardly; they only arise by design. The amazing architectural similarities between executable code and higher genomes clearly indicate that biologists have much to learn from computer scientists, and computer scientists much to learn from biologists.*

Theme 2: Difficulties in Creating Biological Information

There are nine papers included within these proceedings which primarily address the difficulties inherent in creating biological information systems. A thoughtful person who reads the nine papers in the previous section, should immediately see that there are many levels of difficulty.

Intra-cellular communication is essential for life, and involves the continuous flow of information through countless information networks. Any communication network of this type needs many things before it can even begin to function. For example a biological communication network minimally requires at least three material elements: a) *Information senders and information receivers* (in life, these are typically molecules which can both send/receive); b) *Physical media for information transmission* (in life, these are typically messenger molecules); c) *Filtering devices that detect and eliminate false or faulty signals* (in life, DNA repair enzymes, RNases, and proteases). In addition, any biological information network requires at least three non-material

elements: a) *One or more pre-existing languages which can be understood by both senders/receivers* (such language must include vocabulary and grammar – i.e., the genetic code). b) *The actual information to be communicated.* Information can be defined as "that which is communicated". Communicable information is neither the sender-receiver molecules, nor the carrier molecules (e.g., when we send an email, the computers/cables/electrons are just the media, not the message). The information to be communicated is inherently conceptual in nature (it is widely understood that information is neither matter nor energy). c) *Meaning and purpose*. Living communication networks must communicate all the essential specifications for exactly what needs to happen (the meaning) to enable life to be alive (the unifying purpose of all biological information).

So even a simple biological information network cannot begin to operate until the necessary material and non-material components are all in place. In this sense a biological information system is much like a computer system in that both are irreducibly complex - needing all the essential components in place before the system can start. This is most easily seen in a computer system which has many minimal prerequisites - access to an energy network, a hardware system, a software system, one or more languages, and an integrated meaning/purpose hierarchy. In the case of a computer, we know by way of our universal human experience that every single one of those pre-requisites could only arise through the operation of intelligence. For a computer system, it is obvious that all these material and non-material components could never arise spontaneously or fall into place simultaneously. Upon careful consideration, the same should also be obvious in terms of biological information systems. The trial and error process of mutation/selection has no possible relevance to information systems until all the prerequisite components are already firmly in place and operational. Only after an information system is in place is it relevant to ask: "Could mutation/selection improve and expand the established information system?"

Since biological information systems are most like computer networks, it is logical to ask if there are any known self-evolving IT systems. The internet can be viewed as one vast experiment to see if hardware, software, or information systems can self-evolve. So far there is not a trace of evidence of such self-evolution. Everything that is functional within the realm of IT is designed (even the bad stuff). Some might think that computer viruses might arise by an evolutionary process – but they do not; they are all designed. Some computer viruses may be designed to "mutate" in order to evade anti-virus software – but if so, this would just reflect a still higher level of malicious design. It is entirely reasonable to expect that occasionally a random error could (will) enhance a computer virus or a software program. When this does happen, it will merely reflect a trivial and mundane event. It will NOT show that our computers, our software, our internet networks, or our emails generally arise by trial and error. The

entire digital world is extremely powerful evidence that information and information systems only arise by the hard work of countless intelligent designers. The digital world we now live in clearly falsifies the idea that information and information systems can arise by trial and error. Intelligence is coupled to information in a profound way.

Let's continue with the computer analogy. Let's assume that the entire system (the operator, the electrical systems, the hardware systems, the computer language systems, the computer operating systems, and all but one executable program) were already in place. So we just need to create one new executable computer program. Could trial and error do that? This is very similar to the question; "Could mutation/selection ever create a totally new gene within a pre-existing live cell?" The papers that are summarized below show the answer is a resounding "NO".

The first three papers re-examine previous claims that certain software programs have proved that information and information systems can self-evolve. It has been claimed that such programs prove that within a digital environment, the Darwinian mutation/selection process is truly a creative and open-ended process - which can create any amount of new information. It has been widely claimed that when these programs run, they continuously, and without limit, create new information *de novo*. On the basis of these programs, it has been claimed that trial and error might indeed create all the hardware, software, language, and specified meaning/purpose as needed for life. However, these claims can now be strongly refuted.

Tierra: The Character of Adaptation

Winston Ewert, William A. Dembski and Robert J. Marks II

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0005

Dr. Ewert (et al.), who has a PhD in Electrical and Computer Engineering, evaluates the digital-evolution simulation program, Tierra. This simulation was developed by Thomas Ray. Tierra simulates "digital organisms", which are very short programs (short sequences of computer instructions) which were randomly altered within the Tierra simulation. Over time, the sequence of instructions within the small programs changes, and this was termed by Ray "digital evolution". Ray's hope was to demonstrate a digital equivalent of the Cambrian explosion. Initially, Tierra seemed to show some promise, producing a series of "adaptations". However, the few adaptations that arose were very limited in nature and were all dead-ends, leaving Ray and others to deem Tierra a failure.

Dr. Ewert took a closer look as to why Tierra initially showed promise, but eventually failed to deliver anything like a digital Cambrian explosion. He shows that the few Tierra adaptations were due to loss and rearrangement of digital information rather

than creation of new information. When viewed on the genomic level, Tierra "organisms" only underwent trivial or reductive modifications. Simply removing or rearranging existing information has very limited potential for major innovations or creation of new levels of information. For this reason Tierra could only give rise to minor variations, and even such minor alterations only arose for a limited period of time. After a short period of generating a few novel variants, the program always quickly reached a dead-end and stopped "evolving". Dr. Ewert's analysis makes it clear that Tierra was never a genuinely self-creative system, and so failed to produce any meaningful or open-ended evolution.

Significance: *Tierra was not a realistic model of biological evolution, yet it still failed. It teaches us nothing in terms of how real-world biological information networks might be established or expanded. Instead, it only shows us: a) adaptive fine-tuning (small superficial changes in a pre-existing information system); and b) adaptive degeneration (minor adaptations based upon loss of information). It shows a few inherently superficial and limited adaptations - followed by terminal stasis. This is consistent with the study by Basener* (http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0004).

Computational Evolution Experiments Reveal a Net Loss of Genetic Information Despite Selection

Chase W. Nelson and John C. Sanford

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0014

Nelson (and co-author), a PhD candidate in bioinformatics and molecular evolution, thoroughly evaluates the biological implications of the computer program *Avida* (Avida is the program that superseded and replaced the Tierra program described above). Avida was designed to include features that give it the appearance of being more relevant to biological evolution.

Like Tierra, Avida is a computer program which claims to prove "digital evolution", and therefore is thought to prove the feasibility of biological evolution via the mutation/selection process – apart from any design. However, it is important to note that both programs only operate due to enormous amounts of front-loaded design. For example, the Avida program itself was very carefully designed, as were all supporting software programs, all the software languages, all the necessary hardware, power-source systems, etc. Within this vast array of designed systems, was a tiny bubble where "un-directed evolution" might be happening. The only part of the entire information system which was subject to "un-designed change" was a cluster of short strings of computer commands.

As with Tierra, the Avida program begins by establishing a population of many copies of an initially designed "digital organism". This "Avida organism" is just a short string of computer instructions which includes a "self-copy" command. The initial string is just a random string of commands, which contain no information except for the "self-copy command" (which is designed to be immutable). Then the Avida software begins to randomly introduce changes into the Avida organisms (new computer instructions are imported into the string). These instructions are themselves designed units of information. Then the Avida program implements a process of selective competition between the "mutant organisms" to eliminate the *less fit* strings, while allowing only the *more fit* strings to self-copy. "Fitness" in Avida is defined arbitrarily as the ability for a given string of instructions to do certain trivial computational chores (like taking two random numbers and subtracting one from the other). The Avida program then repeats this cycle of mutation, selection, and replication many thousands of times (self-copying, inserting new instructions, and subsequent competitive survival). This highly designed system has some resemblance to biological evolution because it has an element of "mutation" (randomly importing into the string new computer commands), and has an element of competitive selection (by chance some strings can do things that other strings cannot, and these are allowed more opportunity to survive and reproduce).

Nelson's analysis of Avida reveals three serious problems which bring into question the concepts of both digital and biological evolution via the mutation/selection process:

a) Reductive (degenerative) evolution in Avida is extremely strong, as was also observed in Tierra. To circumvent this problem of *genetic entropy*, the Avida designers had to do two things. First, they had to artificially protect Avida's self-copying instructions from mutations (this part of the Avida organism is immutable). In addition, like the designer of Tierra, they had to artificially reward duplication events (otherwise selection for reproductive efficiency favors deletions - which systematically shrink the string of commands and clearly reduce total information).

b) The evolution of traits requiring large numbers of individually-arising bits of information is exceedingly problematic. To circumvent this problem the Avida designers did not even try to modify the Avida string of instructions one binary bit at a time (which would have been more biologically realistic, but would have completely failed to create information). Instead their "mutations" involved adding/subtracting sizable, complete, autonomously-functional units of information (entire instructions). Biologically speaking, this is a little like substituting whole genes instead of making simple nucleotide substitutions. Furthermore, Nelson shows that the Avida designers had to carefully design a smooth stair-step pathway for building Avida's slightly more complex functions. These slightly higher Avida functions were built by combining just a handful of extremely simple lower functions. But even such simple combinations of

instructions were typically too rare to arise spontaneously by themselves (even when letting Avida run tens of thousands of generations). For this reason, just the right intermediate steps had to be designed, with each step arising reasonably frequently, and with each intermediate step being given a very large fitness reward (hence being strongly favored by selection). Nelson shows that the higher Avida functions never evolved when he removed the very strong selection for the purposefully designed intermediate "stepping stone functions". In other words, without the carefully designed stair-step mechanism in place, the modestly higher Avida functions were essentially "irreducible complex". Without this artificially-designed feature, Avida cannot get past the very simplest functions - not even given "deep time". Nelson calls this the *waiting time problem*. This waiting time problem becomes fatal in Avida when more than 7 steps are needed to create a selectable function. This general waiting time problem becomes insurmountable in any evolutionary system when there are very many steps required to create a selectable function. To put this issue in perspective, in real biological systems a minimal functional (selectable) unit of information (a gene), typically involves *thousands* of binary bits of information. The waiting time for such a selectable function to arise spontaneously must exceed the age of the universe.

c) Nelson shows that the problem of selection threshold is deadly to the operation of Avida or any similar system. The designers of Avida made it so that the slightest increase in functionality was given a huge reproductive reward. Likewise, the smallest loss of function resulted in an enormous reproductive penalty. So there was artificially strong selection for beneficial changes and artificially strong selection against deleterious mutations. This was a gross misrepresentation of biological evolution, where most random changes have only very tiny reproductive (selective) consequences.

When Nelson ran the Avida program using more biologically realistic fitness rewards (selection coefficients), Avida's selection mechanism broke down completely. Not even a single one of the simplest functions could evolve. Furthermore, when Nelson initially let Avida run with its artificially high reward settings (so that all of the 9 possible functions arose), and then switched the program to realistic selection coefficients, the Avida organisms underwent systematic degeneration, such that all 9 functions were quickly lost (i.e., the Avida organisms' information content quickly went to zero).

When critically examined by competent biologists, using biologically realistic settings, Avida proves that mutation/selection cannot realistically create a single binary bit of information. In this light we must ask, is there any conceivable way that the entire Avida program could have ever arisen without a designer?

Significance: *Even with massive amounts of front-loaded design, Avida cannot honestly be used to support neo-Darwinian theory. Instead, Avida helps to reveal numerous and profound limitations inherent in the mutation/selection process. Given biologically realistic settings, Avida fails to create a single binary bit of new information. The more we make our genetic simulations biologically realistic, the more clearly the mutation/selection process fails. As will be seen, the most biologically realistic simulation to date (Mendel's Accountant) very clearly reveals the profound limitations of the mutation/selection process.*

A General Theory of Information Cost Incurred by Successful Search

William A. Dembski, Winston Ewert and Robert J. Marks II

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0002

Dr. Dembski (et al.), with PhDs in both Mathematics and Philosophy, is widely known for his work in information theory and information search strategies. Dembski et al. examine in a generic sense, "directed-search programs" (such as Tierra and Avida), which claim to prove that digital mutations/selection can create information. Such programs are designed to purposefully try to seek out instruction strings that can do something (anything). These programs are thought to be relevant to biological evolution because many evolutionists imagine that mutation/selection is itself a naturally occurring "search program". They envision mutation/selection as an un-directed and un-designed "search engine", which can seek out and find life-enabling biological information systems.

Dr. Dembski shows that the reason directed-search engines and genetic algorithms are effective, is because they are intelligently designed. More specifically, they are intelligently designed based upon crucial enabling information available to the program designer. A search program cannot be designed to do any better than a random search, unless the designer has vital information on which to base the search. Without useful information which can guide the search, even a designed search will do no better than random trial and error. For example, the search designer must first have information about what is being searched for. Secondly the search designer must have some information about where the object of the search is most likely to be found. The key operational words for making a search which is superior to a random search are *directed, intelligence, information*. Take away these three elements and you are always back to a random search.

Dr. Dembski uses careful logic and mathematics to show that without information upon which to base a search, even the most brilliant mind cannot design a search which is more effective than random trial and error. If even intelligent designers cannot create effective searches (apart from meaningful guiding information), then obviously neither can un-directed natural forces create a search better than random trial and error. Some

might ask: What if nature could somehow create a *search for a better search*? This rather abstract notion is rigorously examined by Dr. Dembski et al. They show that this intellectual contrivance for creating spontaneous information actually makes the whole problem worse. The time and resources needed to randomly create and test many types of random search strategies, in order to see which is best, will take much more time and resources than a single random search would require.

Significance: *The work of Dr. Dembski et al. indicates that un-directed natural forces could never reasonably be expected to give rise to spontaneous search engines, which could never give rise to spontaneous information, which could never give rise to spontaneous life.*

Limits of Chaos and Progress in Evolutionary Dynamics

William F. Basener

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0004

Dr. Basener, with a PhD in mathematics (specializing in topology and dynamical systems), addresses the problem of biological stasis. He uses mathematics to show that selection is effective in optimizing a biological information system that is already in place, but it cannot lead to a continuous increase of new functions or new information. Instead, what Dr. Basener observed was that what was being selected for quickly reached a natural limit, at which point all advance stopped. By making very minimal assumptions on the evolutionary models, he shows these results hold for an extremely broad class of dynamic models for evolution. He concludes: "Chaos and non-linear dynamics contribute nothing to the on-going increase in complexity or evolutionary fitness of biological systems." He goes on to say: "…the evolutionary process driven by mutation-selection, in both mathematical models and directly observed behavior, is that of a system going to equilibrium and staying there." This is exactly what was observed in the Tierra and Avida programs – a very limited amount of selective progress followed by un-ending stasis.

Dr. Basener's mathematical analysis agrees with the universal experience of biologists - including plant breeders, animal breeders, and lab researchers doing various types of long-term selection experiments. This is also what is seen when adaptation is observed within wild species. Mutations/selection works very well, on a very limited scale. Selective progress is generally very limited in terms of enabling only superficial genetic changes that enable adaptation to a new environment (just fine tuning as opposed to genetic innovation). Once this fine-tuning is achieved, the selective progress stops, followed by stasis.

When plant breeders select for a trait such as fruit size, they initially see rapid progress, but the selective progress quickly approaches a natural limit, leading to stasis. At some

point the very largest fruit have major associated defects (like splitting), and are no longer suitable breeding material. Likewise, when animals are bred for larger size, a natural limit is consistently approached, and the largest animals develop severe pathologies. Similarly, the famous long-term *E. coli* experiment by Lenski et al. shows a relatively superficial adaptation, followed by rapidly diminishing returns. When bacteria were grown for two decades on an artificial diet, the bacteria adapted to the medium, such that growth rate quickly underwent rapid selective improvement. But there is obviously an upper limit in terms of how fast bacteria can grow. Beyond a certain point, on-going selection on the same artificial medium yielded diminishing returns, clearly foreshadowing eventual stasis. In nature it is not unusual to see a species adapt to a new environment. But it is very clear that such adaptation is usually superficial in nature (i.e., a color change, size change, or other simple modification). What we actually observe is that this type of adaptive change seems to always be limited to fine-tuning of pre-existing information (rather than creation of any novel functions), and seems to always quickly lead to rapidly diminishing selective progress and eventual stasis.

Significance: *Strawberries, cows, bacteria, and finches all change in limited and superficial ways (by fine-tuning of existing information), but such minor changes occur only so that a given life form can persist and fundamentally stay the same. Adaptive fine-tuning does not explain the origin of all the underlying information networks which give life to these creatures. Instead, genetic fine-tuning leads only to stasis.*

Multiple Overlapping Genetic Codes Profoundly Reduce the Probability of Beneficial Mutation

George Montañez, Robert J. Marks II, Jorge Fernandez and John C. Sanford

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0006

As mentioned in the previous section, Montañez (et al.), examined the implications of new findings that indicate that within biological information systems there are many overlapping messages and overlapping codes. This represents a very sophisticated form of data compression. Overlapping codes by themselves create major explanatory difficulties: How could they possibly arise by natural process alone? Moreover, because overlapping codes are poly-functional (one DNA sequence can code for two or more messages), they are also poly-constrained (each nucleotide contributes to more than one message - which greatly reduces the chance that any random change could be non-deleterious). The reality of overlapping codes drastically reduces the probability of unambiguously beneficial mutations. This demands that scientists dramatically adjust downward their estimates of the actual rate of beneficial mutation. The mathematical analysis by Montañez et al. indicates that the actual rate of beneficial mutation could easily be 1000-fold less than has previously been thought.

Extremely low rates of beneficial mutation create very serious explanatory difficulties. If unambiguously beneficial mutations are extremely rare, then there are simply too few beneficial mutations to allow for genome-building. For example, the ape-to-man scenario requires that, minimally, tens of millions of beneficial mutations must arise and advance all the way to fixation in just 300,000 generations. This means that hundreds of beneficial mutations must be fixed *every single generation*. Given the analysis by Montañez et al., the beneficial fixation rate would be many orders of magnitude too small to accomplish this. Numerical simulations suggest less than 2,000 beneficial mutations could go to fixation in six million years – even assuming a small population and one beneficial mutation per individual per generation. To make matters worse, it is now well known that a large number of low-impact *deleterious* mutations are continuously accumulating within any natural population, which must lead to unavoidable erosion of information content (http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0010). Traditionally, it has been assumed that such on-going genomic damage might be compensated by on-going amplification of beneficial mutations. But this is not feasible if beneficial mutations are extremely rare. Lastly, only a tiny fraction of the truly beneficial mutations that might arise would be above the population's *selection threshold*. All the other beneficial mutations would be lost due to genetic drift, because they would be too subtle to respond to natural selection. This problem is described in detail below.

Significance: *It is now well established that there are extensive overlapping codes within higher genomes, representing an extremely advanced form of data compression. Deployment of overlapping codes transcends anything computer scientists would even dream of. Overlapping codes represent a quantum leap in our understanding of the sophistication of biological information systems. The Darwinian trial and error mechanism cannot create or improve this type of information technology.*

Selection Threshold Severely Constrains Capture of Beneficial Mutations

John C. Sanford, John R. Baumgardner and Wesley H. Brewer

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0011

Dr. Sanford, with a PhD in plant genetics, along with his two co-authors, demonstrates that there is a fundamental problem when trying to create new biological information via beneficial mutations. Not only are unambiguously beneficial mutations extremely rare (see Montañez et al. above), but the vast majority of such mutations have extremely small biological effects (they are "nearly neutral"). This makes them essentially invisible to natural selection. Dr. Sanford et al. use advanced numerical simulation to show that for any given population and any given set of circumstances, there is a quantifiable *selection threshold*, and any beneficial mutation that has a fitness

effect less than this threshold will not respond to natural selection. Dr. Sanford shows that this phenomenon profoundly limits the capture of beneficial mutations. Under realistic circumstances, only about 1% of beneficial mutations have a strong enough biological effect to be candidates for selective fixation. Therefore, of the few truly beneficial mutations that do arise (see above), the vast majority cannot be selectively amplified, and are lost due to genetic drift. Since most of the functional nucleotides within a higher genome make only a tiny contribution to total biological functionality, most of the genetic information in such a higher organism cannot be attributed to the mutation/selection process.

Sanford et al. go on to explain that the few beneficial mutations that do have substantial fitness effects must necessarily arise independently and in isolation (both in time and in chromosomal location). Since the amount of information that can be attributed to isolated point mutations is extremely limited, the inability to create and select large integrated sets of mutations is extremely problematic from an evolutionary view. There is a huge difference between a point mutation and an integrated, contiguous, ordered set of mutations. It is well known that essentially all information, including essentially all biological information, is context-based. *Any given letter means nothing apart from its context within a much larger array of associated letters*. Hence large numbers of letters must arise simultaneously and in a coherent manner to create meaningful text strings. All of the individual letters in any functional text string (be it DNA, RNA, or protein), are mutually-defining and profoundly inter-dependent. Rare, isolated, high-impact beneficial mutations scattered across the genome can do nothing to create the type of information found within text strings.

Significance: *It has long been thought that since beneficial mutations happen, and since natural selection happens, continuously increasing biological information should be inevitable. This paper shows that this oft-voiced historical perspective was naïve. Not only are there too few beneficial mutations for genome-building, but most of the information in higher genomes is encoded by nucleotides which individually are too subtle to be have been selectively established. The few truly beneficial mutations that arise and have sufficient impact to be selectively amplified only arise independently and in isolation. This profoundly limits their potential impact. They can only accomplish fine-tuning of pre-existing biological information. Realistically, genomes cannot be built one beneficial mutation at a time.*

Explaining Metabolic Innovation: Neo-Darwinism *versus* Design

Douglas D. Axe and Ann K. Gauger

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0022

Drs. Axe and Gauger, both PhD biochemists, examined the many levels of difficulty in trying to explain the origin of the information within metabolic pathways via the Darwinian trial and error mechanism. They identify six levels of difficulty:

a) Cost of Gene Duplication. It is universally understood that neither a protein (hundreds of components), or a gene (thousands of components), can arise spontaneously, regardless of waiting time. So it is assumed that the only way to create new information which might establish a new metabolic enzyme would be via the modification of an already existing protein/gene. This line of thinking presumes that a preexisting gene undergoes accidental duplication, with both of the duplicates continuing to be expressed (transcribed/translated), followed by a series of mutation and selection events. The gene duplication by itself will be deleterious due to disrupted gene regulation, disrupted chromosome architecture, extra DNA replication, and wasted RNA/protein production. Such duplications should be selectively lost, at least until numerous favorable and complimentary beneficial mutations occur in one of the duplicates (but none in the other), in order to create a new function which has enough selective benefit to off-set the costs associated with the duplication. As will be seen below, the waiting time for this to happen under realistic circumstances, even for a single gene, will almost always be prohibitively long.

b) Time to fixation of a beneficial mutation. Almost all new mutations arise and then rapidly go extinct due to random genetic drift. For this reason, in any large population, a beneficial mutation must arise repeatedly, an enormous number of times, before it is NOT lost due to drift. This creates a long waiting time for a given mutation to arise that is not going to be lost. Even when that lucky mutation arises, it still needs a very long time to go to fixation, especially when the selective benefit is small and the population is large.

c) Time to fixation of specific combinations of beneficial mutations. Generally more than a single mutation is required to transform an existing protein into a different protein with a significantly beneficial new function. Generally, a single mutation by itself will have no selectable benefit. If two mutations are required to have a selectable benefit, the waiting time can become seriously prohibitive. In this case the waiting time is much longer because both mutations must be complimentary and must arise almost simultaneously and on the same chromosome. Even when that rare double mutation event finally happens, it will almost universally be lost to drift – until the double mutation has happened repeatedly a vast number of times. When more than two mutations within the same gene are needed to create a selectable benefit, the waiting

time to fixation rapidly becomes prohibitive in the extreme (i.e., greater than the age of the universe).

d) Time to fixation of enough complementary beneficial mutations to establish a significantly new protein. To alter a single protein in a more fundamental way (e.g. to create an entirely new fold), requires many mutually-dependent and mutually-defining beneficial mutations, which must arise upon the same chromosome at more or less the same time. The waiting time for this to happen is staggering. But that set of mutations must occur a vast number of times before the set is NOT lost due to genetic drift.

e) *Time to fixation of enough complementary beneficial mutations to establish a new metabolic pathway.* A metabolic pathway requires many proteins and is affected by many genes. So establishing a new pathway requires a great many beneficial mutations, arising more or less simultaneously in numerous genes and in the same chromosome, all of which are mutually-defining and mutually-dependent. The waiting time is obviously vastly greater than the waiting time for just a handful of mutually dependent mutations within a single gene.

f) *Causal Circularity*. There are numerous biosynthetic pathways where the molecule being made is required for the pathway to operate that produces it. This is a little like the old adage "you have to have money to make money". Viewed on a larger scale, all of the essential components of a cell are like this – each essential component is necessary for the other essential components to be synthesized and functioning. So ultimately each such component is needed for its own synthesis. So how did that gene get established originally?

Significance: *The authors make it very clear that metabolic pathways cannot be created one mutation at a time. Their last point might be expressed most broadly in the form of a new adage: "To make any one of the essential components of life, one must already have that component present - plus all the other essential components of life". I believe this is the fullest expression of the concept of biological irreducible complexity.*

Supplemental Papers on Self Organization. In addition to the three original sections presented at the Cornell symposium, a fourth section is added to these published proceedings, incorporating the work of Drs. Kauffman and Weber (edited by Dr. B. Gordon). These supplemental papers focus on the concept of "self-organization". The self-organization model proposes that while the standard neo-Darwinian theory is not necessarily wrong, by itself it is insufficient to explain life and the generation of biological information. The self-organization thesis is that inherent in natural reality is the ability for complex systems to arise spontaneously, even apart from the

mutation/selection process. Note: These two papers have a combined "significance" section.

A) Evolution Beyond Entailing Law: The Roles of Embodied Information and Self Organization

Stuart Kauffman

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0023

Dr. Kauffman is currently Distinguished Professor of Biochemistry and Mathematics at the University of Vermont and Distinguished Professor of Computational Systems Biology at the Tampere University of Technology in Finland. Dr. Kauffman is also a well-known theorist and advocate of self-organization. Dr. Kauffman discusses his own model of life, wherein the origin of life and biological information is inherently non-deterministic. He therefore contends that "physics ends where life begins". He similarly argues that since life is non-deterministic, the concept of front-loaded intelligent design is not a viable source of biological information. He argues that life is not predicted by any specific set of natural laws. Rather it *is in the very nature of nature* that "life bubbles forth". Dr. Kauffman asserts there is "a natural magic, creativity beyond the entailing laws of modern physics." He goes on to assert that the evolving biosphere literally constructs, without selection, its own future possibilities. Dr. Kauffman urges that this framework be examined in more depth.

See next summary for significance discussion…

B) Towards a General Biology: Emergence of Life and Information from the Perspective of Complex Systems Dynamics

Bruce H. Weber

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0024

Dr. Weber, Professor of Biochemistry at California State University Fullerton and at Bennington College, is also a well-known theorist and advocate of self-organization. Dr. Weber presents an overview of the self-organization perspective. He describes the tension between the reductionist view (biological reality is found in the components of life and their predictable interactions, with higher level life systems being merely "epiphenomena"), versus the holism perspective (biological reality is found in the organism, its molecular components are merely epiphenomena). Dr. Weber holds to the holism view, that life is more than its components/interactions. Like Dr. Kauffman, he does not feel this points to any type of intelligence, but rather contends that it is in the very nature of nature that higher levels of organization spontaneously emerge from lower levels, and furthermore such emergence is not dependent on natural selection. Rather, natural

selection itself is an emergent phenomenon. Dr. Weber gives three examples of the basic three levels of emergence: 1) The liquid properties of water emerge from the molecular nature of water; 2) Convection cells emerge from the thermodynamic disequilibrium of bottom-heated fluids (related examples – snowflakes, tornadoes); and 3) evolving life systems emerge from storable information which specifies survival. On this last point Dr. Weber states "The hard problem in origin of life research is… how it came to be that a digital-type code in nucleic acids came to specify the analogical information in the thousands of proteins that catalyze metabolism and are involved in signally and processing…" It is on this last point that Drs. Kauffman and Weber come into agreement with the other 27 scientists contributing to this volume. The neo-Darwinian mechanism of mutation/selection can neither explain the origin of biological information nor the origin of biological information systems. Stating this more broadly - a purely reductionist (Darwinian) explanation for the origin and maintenance of all of life, including mankind, is no longer credible.

Significance: *Most of the papers presented in this book have been research papers which presented detailed scientific analyses of specific scientific issues. Symposium authors were asked to stick to their scientific analysis and at most, to only touch on philosophical issues in passing. However the authors of these last two supplemental papers were given greater license, and so provided essays that are primarily philosophical in character. These papers were welcome additions to this book. They broaden the range of presented "new perspectives". These two authors oppose the concept of "intelligent design", but I take the liberty to point out that they also oppose the strictly materialist explanation. Dr. Kauffman suggests that information systems arise via what he calls "natural magic". For me personally, this seems to presuppose a type of magic that requires some kind of intelligent magician. Similarly, Dr. Weber suggests that the natural world has built into it the natural ability (and apparently the inclination) to spontaneously organize itself into highly ordered information systems (such as living cells). If this is indeed true, such a remarkable built-in ability and inclination requires a cogent explanation. In my mind it strongly points to an intelligent cause.*

Theme 3: Difficulties in Preventing Erosion of Biological Information

Eight of the papers included within these proceedings primarily investigated the problem of loss of biological information. Loss of information is something we can all

understand - it is part of our everyday experience. Like our own human information systems, the elaborate information networks within a cell can be disrupted by countless random events. Most fundamentally, biological information is disrupted by random mutations. Mutations are the cause of hereditary diseases, cancers, and immeasurable heartache. In fact, mutation is the primary underlying cause of aging and death. Since it is well known that deleterious mutations can accumulate even when there is strong selection, and since beneficial mutations are very rare, it appears highly problematic: How could the mutation/selection process result in a *net gain* of information over time? In this last part of the synopsis, we examine the problem of genetic entropy, and we ask if Darwinian selection can effectively halt genetic degeneration. If natural selection cannot preserve biological information through deep time, we clearly need to explore alternative models of how biological information arises.

Getting There First: An Evolutionary Rate Advantage for Adaptive Loss-of-Function Mutations

Michael J. Behe

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0020

Dr. Behe, an expert in cell biology and biochemistry, shows that all "beneficial" bacterial mutations which he has analyzed consistently turn out to entail a loss-of-function rather than creation of a new biological function. Loss of function mutations involve such things as deleted genes, inactivated genes, or otherwise disrupted genes. This means that even useful adaptive mutations usually still involve a net loss of information. Dr. Behe makes a theoretical analysis of why this should be. He shows that when a bacterial population encounters a new unfavorable environment, there is *urgency* in dealing with the new challenge (or else the population will go extinct). This means that selection does not have time to find the *best* mutation for solving the problem, but will consistently amplify the *first* mutation that resolves the immediate need (even when the change is deleterious in the big picture). Loss-of-function mutations are vastly more common than gain-of-function mutations (picture introducing typographical mistakes into an instruction manual). Therefore most adaptive selection events will amplify loss-of-function-mutations (they almost always arrive on the scene first). Because loss-of-function mutations inherently involve loss of information, there tends to be a net loss of information even while meaningful adaptation is happening.

Significance: *This paper shows that even when adaptive mutations do happen, they will almost always be manifested as a loss of functional information. This is because, given a pressing environmental challenge, selection will favor whatever solution to the problem arises first. Since there are many ways to break a gene, but very few ways to make a gene better, the first solution to arise will almost always involve a loss of functional information. Dr. Behe's theoretical analysis is in perfect agreement with his*

previous analyses showing empirically that real-world adaptive mutations consistently involve loss of information. This paper, stands alongside the paper by Dr. Montañez et al. (Multiple Overlapping Genetic Codes Profoundly Reduce the Probability of Beneficial Mutation, http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0006) *both papers show that beneficial mutations are almost always only beneficial in a narrow or superficial sense, but in the bigger picture are consistently degenerative in nature, in terms of information content.*

Can Purifying Selection Preserve Biological Information?

Paul Gibson, John R. Baumgardner, Wesley H. Brewer and John C. Sanford

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0010

Dr. Gibson (et al.), a plant geneticist, demonstrates an even more fundamental problem regarding net loss of information. It is obvious that within the functional genome most mutations should be deleterious (they destroy information), while vanishingly few mutations should be beneficial (severely limiting creation of additional information). It is often claimed that deleterious mutations do not matter, as natural selection removes them. But this is clearly not correct. Deleterious mutations generally cannot be removed via selection, because most of them have very tiny biological effects. As a result, most deleterious mutations are too small to be recognized by natural selection and so accumulate continuously, like rust on a car. This basic problem has been known by population geneticists for a very long time (sometimes it has been called "the near-neutral mutation problem" or "the genetic load problem"). Gibson et al. have gone far beyond previous studies, and show that the problem is much more severe than previously recognized. Their findings show that, in higher organisms, the large majority of deleterious mutations are too subtle to be selected away. Consequently, deleterious mutations should systematically destroy biological information. Natural selection can slow down, but cannot stop, this degenerative process. Selection should only eliminate the worst mutations. Unless there is some unknown mechanism which can eliminate huge numbers of deleterious mutations which have tiny biological effects, most deleterious mutations must accumulate continuously, resulting in a continuous and progressive loss of biological information.

Significance: *This fundamental theoretical problem of deleterious mutation accumulation is not new, but it has been consistently clouded by confusion. This paper uses a new method of analysis (comprehensive numerical simulation), which finally brings clarity to the issue. It is now abundantly clear that the deleterious mutation accumulation problem is very real, and in fact is much more serious than has previously been thought.*

Using Numerical Simulation to Test the "Mutation-Count" Hypothesis

Wesley H. Brewer, John R. Baumgardner and John C. Sanford

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0012

Some have argued that the long-standing problem of continuous accumulation of deleterious mutations might theoretically be solved if natural selection could be focused on the elimination of those individuals with the most accumulated mutations. Dr. Brewer (et al.), is a computer scientist with expertise in numerical simulation. Brewer et al. show that this mechanism only works under extremely unrealistic biological conditions, and shows that this artificial mechanism should be completely ineffective in the real world.

Significance: *The theoretical problem of accumulating deleterious mutations has often been dismissed by invoking mechanisms wherein selection eliminates the individuals with the most numerous mutations. This paper effectively falsifies this hypothetical mutation-count mechanism, leaving the problem of deleterious mutation accumulation un-resolved, and leaving the neo-Darwinian mechanism without a credible defense.*

Can Synergistic Epistasis Halt Mutation Accumulation? Results from Numerical Simulation

John R. Baumgardner, Wesley H. Brewer and John C. Sanford

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0013

Some have proposed that the problem of continuous accumulation of deleterious mutations might be solved if natural selection could be used to eliminate individuals with the most "synergistic epistasis". Dr. Baumgardner is a computer scientist with expertise in numerical simulation. Dr. Baumgardner et al. show that this theoretical mechanism is highly contrived and is not even remotely realistic. He then goes on to show that even if there were pervasive synergistic epistatic interaction (it is a rare deviation from normal gene interactions), it does not solve the deleterious mutation accumulation problem, but only makes the information degeneration problem worse.

Significance: *Synergistic epistasis is a rare deviation from normal genic interactions, and it would never even be discussed, except that it has been invoked as a solution to the mutation accumulation problem. The mechanism has been largely as an abstraction, as a way to dismiss the mutation accumulation problem. Synergistic epistasis, as it would apply on a genomic level, has never been rigorously examined. Dr. Baumgardner et al. for the first time rigorously examine the hypothesis that synergistic epistatic interactions might solve the mutation accumulation problem on the genomic level. The authors effectively falsify the hypothesis, leaving the problem of deleterious mutation accumulation un-resolved, and again leaving neo-Darwinian theory without an effective defense.*

Computational Evolution Experiments Reveal a Net Loss of Genetic Information Despite Selection

Chase W. Nelson and John C. Sanford

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0014

As discussed in the prior section, the computer program "Avida" is claimed to be literally a type of "digital life". This program has been used to try and show that selection can be effective in eliminating deleterious mutations even while amplifying beneficial mutations. Unfortunately, Avida's default settings do not reflect biological reality. For example, all Avida mutations are designed to have enormous biological effects (geneticists know that just the opposite is true, most mutations have tiny effects). Nelson (and co-author) shows that when Avida mutations are assigned realistic biological effects, the program fails to amplify any beneficial mutations. More relevant to this discussion, Avida completely fails to halt the continuous loss of information already present. With realistic parameter settings, the Avida population systematically loses all established information. It does not go extinct, but only because Avida was designed to continue to operate even when there is no more information left to lose. The evolutionary Avida program, given biologically realistic parameter settings, provides compelling evidence that low impact mutations are un-selectable. Avida strongly confirms that continuous loss of information is a very real problem, strongly indicating that the mutation/selection process is not sufficient to stop on-going net loss of information.

Significance: *Avida is a life-simulation computer program which many have claimed proves that the Darwinian mechanism is effective at creating a net gain in information. It appears to do this by eliminating all deleterious mutations and simultaneously amplifying all beneficial mutations. But when Avida is run using biologically realistic parameters, what is seen is just the opposite. There is a consistent net loss of information (to the point where all information which is subject to mutation is lost), because low-impact deleterious mutations consistently escape purifying selection. Beneficial mutations fail to accumulate. To the extent that Avida reflects the Darwinian process, it very effectively falsifies neo-Darwinian theory.*

Information Loss: Potential for Accelerating Natural Genetic Attenuation of RNA Viruses

Wesley H. Brewer, Franzine D. Smith and John C. Sanford

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0015

Dr. Brewer (et al.) uses his expertise in numerical simulation to show that loss of information is not always a bad thing. RNA viruses have a high rate of mutation, and are known to be prone to mutational degeneration. Dr. Brewer et al. show that erosion of information due to mutation accumulation probably plays a significant role in

extinction of viral strains and the cessation of pandemics. They further show that pharmaceuticals that increase RNA mutation rates should be highly effective in reducing severity and duration of pandemics. These theoretical studies have been subsequently validated by an historical analysis of the human H1N1 strain of influenza. Since the pandemic of 1918, that influenza strain underwent continuous accumulation of mutations at a very constant rate, such that over 10% of its genome became mutated. During the same time, the strain underwent continuous and dramatic attenuation (as evidenced by reduced pathogenicity), and in 2009 the human H1N1 strain apparently went extinct (see - A New Look at an Old Virus: Pattern of Mutation Accumulation in the Human H1N1 Influenza Virus Since 1918 http://www.tbiomed.com/content/pdf/1742-4682-9-42.pdf).

Significance: *It has often been said that viruses in general, and specifically influenza, are proof that the mutation/selection process creates new information. This study shows just the opposite. Viral strains can certainly undergo fine-tuning in terms of adaption to their host or adaption to antiviral pharmaceuticals. However, RNA viruses such as influenza are inherently subject to spontaneous degeneration due to deleterious mutation accumulation. This can lead to genetic degeneration as reflected by attenuation of its affects, pandemic termination, and strain extinction.*

Entropy, Evolution, and Open Systems

Granville Sewell

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0007

Dr. Sewell, a mathematician, examines the relationship between biological information and the principles of thermodynamics. He falsifies the commonly asserted claim that all that is needed for the creation, maintenance, and expansion of life and biological information is "an open system" with an external energy source. It is universally understood that entropy (disorder) cannot decrease in an isolated system (the system does not self-order itself). But our earth is an open system, and so entropy might decrease (order can increase), as long as there is a compensating increase (more disorder), outside the earth. This common argument asserts that the spectacular decrease in entropy seen on earth (i.e., associated with life, computers, etc.), is compensated by increases in entropy of the sun. Sewell challenges this compensation idea by showing that in an open system, the "X-entropy" associated with any diffusing component X (if X=heat, X-entropy is just thermal entropy) cannot decrease faster than it is exported through the boundary. Stated another way, the X-order in an open system cannot increase faster than it is imported. Thus, he argues, the very equations of entropic change, upon which the compensation idea is based, when they are examined more closely, actually support the common sense conclusion that "if an increase in order is extremely improbable when a system is isolated, it is still

extremely improbable when the system is open, unless something is entering which makes it NOT extremely improbable."

Significance: *In terms of direct observation, it is our universal experience that the only meaningful counterforce to entropic degeneration is an intelligent will. This is the underlying factor which allows people, human society, and life itself - to resist entropic decay. Picture a young lady's bedroom, which has been undergoing increasing entropy (it is a mess). When it is a closed system (with nothing entering or leaving), the room will never organize or clean itself. But what if it is an open system (so things can enter or leave)? For example, what if we import energy? Will turning up the thermostat reduce the room's disorder? Will letting sunlight in through the window reduce the disorder? Will opening the widow let disorder escape? What might come in through the window that might reverse the entropy? Letting birds and insects in will not organize the room. Dr. Sewell points out that whatever is impossible within a closed system (i.e., a room that might self-organize), is on a practical level still impossible in an open system. The only thing that can come into the room and reverse the disorder would be an intelligent agent (i.e., the young lady), or an agent of intelligence (a housekeeping robot). Only an intelligent will can reverse the growing entropy in the room.*

Information and Thermodynamics in Living Systems

Andy C. McIntosh

http://www.worldscientific.com/doi/pdf/10.1142/9789814508728_0008

Dr. McIntosh, an expert in thermodynamics, investigates the relationship between biological information and the principles of thermodynamics applied to open systems. He briefly describes the second law of thermodynamics which says that in isolated systems the amount of useful energy (which can do work), is always decreasing. He then moves into the main thesis of his paper, which is that even in a non-isolated system there are crucial thermodynamic principles that apply. In a system such as the earth's biosphere (constantly receiving energy from the sun) the second law does not strictly apply, causing some to posit that by adding energy to the system, one should be able to reverse the overall trend of entropic degeneration and enable the spontaneous development of life with all its nano-machines and biological information systems. Dr. McIntosh critically examines this widely-held idea among biologists that all one needs is an external energy source in order to discard the fundamental problem of thermodynamic decay and entropic degeneration.

Dr. McIntosh shows with a number of examples that machinery (defined as devices which harness energy from an external source and use it to do work) never arise just by simply bringing random energy across the open boundary of a system. Such free energy (energy available to do work) requires there to have already been a mechanism

in place ready to capture and convert such incoming energy. So the sun's radiant energy, by itself, does nothing to produce or enable the origin of biological machinery. The opposite is true – biological machinery (i.e., a plant's photosynthetic machinery) enables the capture/channeling of energy to make sugars to allow plant growth and biosphere development. Information is needed to make the machinery, and without the machinery the sun's energy can do no useful work. Without functional information (specifications), the external energy source is biologically useless. Without information (i.e., intelligence), the external energy source does nothing to resolve the problem of thermodynamic constraint on self-organization, or the problem of entropic decay.

Dr. McIntosh then goes on to show a second very important issue that emerges from this. The information machinery of DNA/ribosome/protein manufacture etc. in all living systems is itself sitting on a free energy substrate. In other words, the DNA itself is formed with disequilibrium thermodynamics being sustained – there is an 'uphill' thermodynamic process going on. The polymerization of DNA itself requires precise energy input to make the joining and stringing together of DNA to take place. The same is also true for the proteins encoded by DNA.

This led Dr. McIntosh to a third important observation. He asked: What is it that constrains the thermodynamics to be in such a disequilibrium? To answer this, one has to address the question: What is information? Surprisingly, it is NOT the DNA chemistry that makes information. The information is defined by a *code* (similar to the software of a computer). It is the *arrangement* of the chemicals which is used by the messaging system of DNA, and this underlying information is *non-material*. It is NOT the thermodynamics of the matter and chemical/electrical energy which determines information content, but rather the reverse. It is *the very presence of the informational logic (i.e., coded information), which constrains the thermodynamics* and orders the matter and energy in the system, using and controlling free energy devices (machines). This is a fundamental conclusion and has far reaching consequences for understanding biological systems.

Dr. McIntosh addresses a fourth issue that arises out of his study. He proposes several principles of (non-material) coded information exchange. These principles mirror some of the laws of thermodynamics. He then moves on to propose principles concerning how non-material information interacts with matter and energy in (open) biological systems. The crucial conclusion is what he refers to as the 'top down' principle: *information enables bio-machinery to work, which enables the capture of energy and thus raises the free energy within the system.*

In terms of practical application, Dr. McIntosh shows that what allows life to be alive is not biochemistry plus an external energy source. Rather, life is possible because of *biological information* – which allows capture of external energy, directed energy

processing and enhancement, and the use of such energy to build and run molecular machinery along with a multitude of metabolic networks. Such energy-rich molecular machines and networks are themselves specified and controlled by biological information. *The molecules, machines, and networks do not make the information. Rather, the information makes the molecules, machines, and networks.* The neo-Darwinian view has always been just the opposite - organic molecules plus energy give rise to information. Dr. McIntosh proposes that information is primary, while molecules are secondary.

Significance: *It is sometimes incorrectly stated that life violates the second law. This is not correct and creates confusion, because living systems are not isolated. There is always an external energy source. But this is not where the Darwinian mechanism fails. The fallacy is in the assertion that energy on its own can build the necessary machinery of life. This does not occur and cannot occur thermodynamically. Science repeatedly shows this not to be the case. However, because life involves many layers of intricate coded and nested software programs, life does something very extraordinary – it actively resists going to its lowest energy state. Life has the unique ability to "hover", in a sustained manner, far above the energy state of an otherwise dead or decaying organism. This happens specifically because it has coded information instructions which actively capture and channel the energy available, for necessary synthesis, repair and maintenance of all systems. In this way life can remain in a suspended state of extreme disequilibrium.*

This can be visualized nicely by considering a hovering hummingbird. It does not go to its lowest available energy state (on the ground - dead and decaying), but instead maintains itself in an exceedingly improbable state of disequilibrium. This is possible, in part, because within the nectar which the bird drinks there is more than enough metabolic energy for that needed for the bird to hover. But that is not the interesting part. High-quality raw energy by itself is NOT what really makes the hummingbird hover. It is necessary but not sufficient. It is only the bird's very high quality biological information that channels the available energy in precisely the right way which enables and maintains the bird's perfect levitation. The required information is resident in the bird's brain, nervous system, muscles, feathers, hollow bones, cells, proteins, ATP synthase, RNAs, and DNAs. This information is not just a series of zeros and ones floating around somewhere within the bird. The information is active and "alive" within a labyrinth of information networks. These networks require a vast matrix of senders and receivers, as well as many languages, and massive global integration. Every component of every cell, within every tissue, within every organ of the bird, requires continuous information flow. The biological information which levitates the hummingbird is the collective effect of the operation and interaction of countless executable programs.

Based upon everything we know about information systems, this biological labyrinth of information systems, which is required for the life to be alive, clearly seems to be the outworking of a fundamental underlying intelligence. It should be obvious to any biologist that this amazing information labyrinth is what enables the hummingbird to hover and be alive. The only rational basis for the existence of such an information network is some type of underlying intelligence. The reason why so many biologists vehemently deny this obvious conclusion is their unwavering philosophical commitment to strict materialism.

The flowering plant, from which the hummingbird obtains its energy, has its own enabling labyrinth of information which allows it to photosynthesize and grow. The plant has no brain, yet its enabling information labyrinth also appears to be the outworking of a fundamental underlying intelligence. Reasonably, it is this underlying intelligence that enables the information labyrinth, which enables the plant to capture low-quality radiant energy from the sun, and convert it into higher quality chemical energy (that the machinery in the hummingbird can use to do work), which enables the hummingbird to hover. The sun provides the energy, but biological information is the basis for capturing the energy, improving its form and quality, and directing it to create, maintain, and operate the machinery needed for life. It is information that enables life to intelligently control and make use of the downward flow of energy (thermodynamics).

Figures on next page: The hovering hummingbird, along with the flowering plant that nourishes it, epitomize life's amazing ability to persist in sustained thermodynamic disequilibrium (top image). Many factors are involved, but the primary "vital force" which enables hummingbirds and plants to "hover" far above thermodynamic equilibrium, is active biological information flowing continuously through elaborate information channels (bottom image).

Final Comments from the Author

When I first conceived of the Cornell symposium in 2010, I could not have imagined that it would attract so many gifted scientists from so many diverse disciplines. It was my privilege to work with Drs. Marks, Behe, Dembski, and Gordon in enlisting the speakers, getting the papers reviewed, and editing the proceedings. I believe everyone who contributed to the symposium went away with a greatly enhanced appreciation of what biological information really is (certainly I did). When you start to see it, the depth and sophistication of biological information is simply breathtaking.

Many scientists who are committed to the standard neo-Darwinian model of life may find these proceedings disturbing – which is unfortunate. I do not think any of the contributing authors to the proceedings had any intention to offend anyone. It is just that it is increasingly clear that the long-reigning neo-Darwinian paradigm is collapsing – and despite many efforts to deny what is obvious – clearly "the emperor has no clothes." The extremely sophisticated hardware and software systems that enable life simply cannot be built by any trial and error system. In particular – it is very clear that software can never be developed one binary bit at a time. Apart from a fully functional pre-existing hardware/software system, a single bit has absolutely no meaning. I feel that if we are to preserve our scientific integrity, we must acknowledge that we have a major explanatory problem, and we need to go back to the drawing board in terms of understanding the origin of biological information.

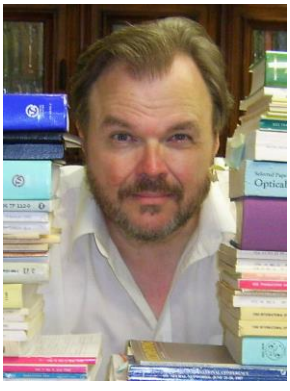
The entropic degeneration of information is something we all understand – it is a general problem we all have to deal with every day. It is clear that this is also an enormous problem within the biological realm. We all have a limited life expectancy – primarily due to mutation accumulation on the personal level. The problem of mutation accumulation is clearly also a serious problem on the level of the species. Selection does not generally appear to be capable of halting deleterious mutation accumulation, and most genetic adaptations appear to involve loss of information. The problem of entropic degeneration of biological information should not be swept under the rug. While it seems paradoxical within the ruling paradigm, it is extremely important and clearly deserves to be studied in much more depth.

J. C. Sanford

Biographies – Authors/Editors

Robert J. Marks II

Distinguished Professor, Baylor University, USA



Robert J. Marks II is currently the Distinguished Professor of Electrical & Computer Engineering at Baylor University. He is the author, coauthor, Editor, or Coeditor of eight books published by MIT Press, IEEE, and Springer-Verlag. His most recent text is Handbook of Fourier Analysis and Its Applications (Oxford University Press, 2009). His research has been funded by organizations such as the National Science Foundation, General Electric, Southern California Edison, Electric Power Research Institute, the Air Force Office of Scientific Research, the Office of Naval Research, the Whitaker Foundation, Boeing Defense, the National Institutes of Health, The Jet Propulsion Laboratory, the Army Research Office, and the National Aeronautics and Space Administration (NASA). Dr. Marks is Fellow of the IEEE and the Optical Society of America. He is a former Editor-in-Chief of the IEEE TRANSACTIONS ON NEURAL NETWORKS. He was the recipient of numerous professional awards, including a NASA Tech Brief Award and a Best Paper Award from the American Brachytherapy Society for prostate-cancer research. He was the recipient of the Banned Item of the Year Award from the Discovery Institute and a recognition crystal from the International Joint Conference on Neural Networks for contributions to the field of neural networks (2007).

Werner Gitt

German Federal Institute of Physics and Technology. Former Director and Head of Information Technology.

Dr. Gitt obtained an undergraduate degree in engineering from the Technical University of Hannover in 1968 and completed his Ph.D. summa cum laude in 1970 from the Technical University of Aachen which also awarded him its prestigious “Borchers Medal.” In 1971 Werner Gitt started his career at the German Federal Institute of Physics and Technology in Brunswick, being promoted to Director and Professor in 1978. He served as Head of “Information Technology” from 1971 to 2002, when he retired. He is the author of numerous research papers dealing with information science, numerical mathematics, and control engineering.

William A. Dembski
Discovery Institute, 208 Columbia Street, Seattle, WA 98104.

William A. Dembski received a B.A. degree in psychology, a M.S. degree in statistics, a Ph.D. degree in philosophy, and a Ph.D. degree in mathematics in 1988 from the University of Chicago, Chicago, IL, and the M.Div. degree from Princeton Theological Seminary, Princeton, NJ, in 1996. He was an Associate Research Professor with the Conceptual Foundations of Science, Baylor University, Waco, TX. He is currently also a Senior Fellow with the Center for Science and Culture, Discovery Institute, Seattle, WA. He has held National Science Foundation graduate and postdoctoral fellowships. He has published articles in mathematics, philosophy, and theology journals and is the author/editor of more than a dozen books.

John W. Oller, Jr.
Hawthorne Regents Professor IV, Department of Communicative Disorders, University of Louisiana at Lafayette

John W. Oller, Jr., Ph.D. founded the Department of Linguistics at the University of New Mexico in 1972 and the Applied Language and Speech Sciences Ph.D Program at UL Lafayette in 2001. Oller's research has concentrated on the theory and experimental measurement of linguistic processes in education, high stakes testing, the diagnosis of disorders, the success of social interactions, and more recently on genetic systems, biochemistry, repair and disease defenses, etc. Winner of the Mildenberger Prize offered by the Modern Language Association, Oller is the author of over 200 peer-reviewed papers and monographs along with 16 books largely in experimental measurement and research on theories of linguistics and sign systems in general. His 2010 works include a book on the causes of autism, an encyclopedic reclassification of communication disorders and related disease conditions, and a monograph-sized contribution to the peer-reviewed multidisciplinary open source journal Entropy. The latter deals with the process of pragmatic mapping (as in referring to an object, person, event, relation, or sequence of them) and as found in genetics, the dynamics of immune systems, and the distinct neuroarchitecture of the human brain.

William F. Basener
School of Mathematics, Rochester Institute of Technology

Dr. Basener is an associate professor in the School of Mathematical Sciences at the Rochester Institute of Technology and Chief Imaging Scientist for Spectral Solutions. He received a bachelor's degree in mathematics from Marist College and a Ph.D. in mathematics from Boston University in 2000. He has published research in dynamical systems, chaos, topology, population modeling, economics and remote sensing and is the author of an NSF-funded textbook, *Topology and Its Applications*. He has also worked on projects funded by the Dept. of Defense, various corporations, and has worked as a contractor for the National Geospatial-Intelligence Agency.

Winston Ewert
Electrical & Computer Engineering, One Bear Place #97356, Baylor University, Waco, TX 76798-7356

Winston Ewert received a B.Sc. in Computer Science from Trinity Western University in Langley, B.C., and a Ph.D. at Baylor University where he was a member of Evolutionary Informatics Lab. Together with Dr. Robert J. Marks II, Dr. William Dembski, and George Montañez, he is an author on a number of papers investigating the informational content of evolution-inspired search algorithms. He now works as a Software Engineer.

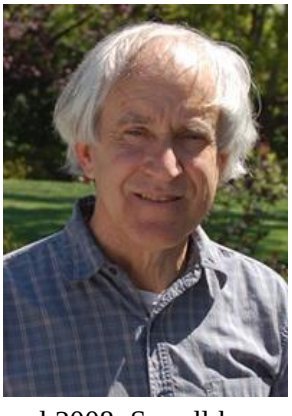
George D. Montañez

BS Computer Science, University of California –Riverside (2004), MS Computer Science, Baylor University (2011)

George D. Montañez is a graduate student in the Machine Learning department, School of Computer Science, at Carnegie Mellon University. His research interests include predictive state model reconstruction, information properties of genetic algorithms, conservation of information in machine learning, and machine learning methods for textual data mining. He served as a research assistant to Dr. Robert J. Marks II at Baylor University.

Granville Sewell

Mathematics Department, University of Texas, El Paso.



Granville Sewell is Professor of Mathematics at the University of Texas at El Paso (UTEP). He completed his Ph.D in Mathematics at Purdue University, and has subsequently been employed by (in chronological order) Universidad Simon Bolivar (Caracas), Oak Ridge National Laboratory, Purdue University, IMSL Inc. (Houston), UTEP, The University of Texas Center for High Performance Computing (Austin), and Texas A&M University, and is currently back at UTEP. He spent one semester (Fall 1999) teaching at Universidad Nacional de Tucuman in Argentina, on a Fulbright grant, and returned to Universidad Simon Bolivar to teach summer courses in 2005 and 2008. Sewell has written three books on numerical analysis, and is the author of a widely-used finite element computer program (video at www.roguewave.com/pde2d).

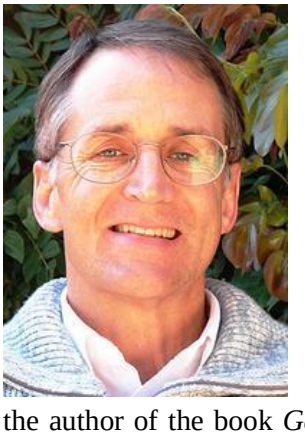
Andy C. McIntosh

Prof. University of Leeds, DSc, FIMA, C.Math, FInstE, CEng, FInstP, MIGEM, FRAeS

Andy McIntosh holds a research chair in Thermodynamics and Combustion Theory, and has lectured and researched in these fields for over 30 years. He has a Ph.D. in combustion theory from the aerodynamics department of what was then Cranfield Institute of Technology (now Cranfield University), a DSc in Applied Mathematics from the University of Wales and worked for a number of years at the Royal Aircraft Establishment. He is a Fellow of the Institute of Mathematics and its Applications, the Institute of Energy, the Institute of Physics and the Royal Aeronautical Society. A chartered mathematician and engineer, and author of over 180 papers and articles, his research has been in combustion in fluids and solids. His work has also included investigations into the fundamental link between thermodynamics and information, and in the last few years he has been involved in research in the area of biomimetics where the minute combustion chamber of the bombardier beetle has inspired a patented novel spray technology with applications to fuel injectors, pharmaceutical sprays, fire extinguishers and aerosols. This research was awarded the 2010 Times Higher Educational award for the Outstanding Contribution to Innovation and Technology.

John C. Sanford

Department of Horticulture, NYSAES, Cornell University, Geneva NY 14456



John Sanford has a Ph.D. in Plant Breeding/Genetics from the University of Wisconsin. He has been a Cornell professor for over 30 years, conducting research in the areas of plant breeding, plant genetic engineering, and theoretical genetics. John conducted plant genetic research that resulted in many new crop varieties, more than 100 scientific publications, and several dozen patents. John was the primary inventor of the biolistic "gene gun" process, which was used to produce a large fraction of the transgenic crops grown in the world today. John was team leader in the development of the program Mendel's Accountant, the world's first biologically realistic forward time genetic accounting program. John is the author of the book *Genetic Entropy and the Mystery of the Genome*. John is now semi-retired from Cornell, and continues to hold the position of Courtesy Associate Professor.

Jonathan Wells

Discovery Institute, Seattle WA 98104.

Jonathan Wells holds an A.B. in Physical Sciences from the University of California at Berkeley. In 1985 he received a Ph.D. in Religious Studies from Yale University, with a dissertation on Charles Hodge and the nineteenth-century Darwinian controversies. In 1994 he received a second Ph.D. in Molecular and Cell Biology from the University of California at Berkeley, with a dissertation on frog embryology. From 1995 to 1998 he worked as a hospital laboratory supervisor and did postdoctoral research at Berkeley. He then moved with his family to Seattle, where he is now a Senior Research Fellow at the Discovery Institute. He has authored scientific articles in BioSystems, The Scientist, The American Biology Teacher and Rivista di Biologia / Biology Review, and he has co-authored articles in Development and Proceedings of the National Academy of Sciences USA. He is also the author of several books, including *Charles Hodge's Critique of Darwinism*, *Icons of Evolution* and *The Politically Incorrect Guide to Darwinism and Intelligent Design*, and he is the co-author (with William Dembski) of *The Design of Life*. His most recent book, *The Myth of Junk DNA*, was published in 2011.

Paul Gibson

Adjunct Associate Professor, Dept. of Plant, Soil, and Agricultural Systems, Southern Illinois University. Professor, Plant Genetics and Statistics, Cooperative Studies, Inc., Overland Park, KS.

Paul Gibson has had a career-long interest in theoretical quantitative genetics and its application to plant breeding for the improvement of food crops in hungry areas of the world. His Ph.D is in Plant Breeding and Cytogenetics from Iowa State University in 1981, with his dissertation research conducted at the International Crops Research Institute (ICRISAT) in India. After working as a maize breeder in Zambia, he conducted quantitative genetic and molecular research and taught at Southern Illinois University. Paul now serves as the primary instructor and mentor in a regional MSc and Ph.D program in Plant Breeding and Biotechnology at Makerere Univ. in Kampala, Uganda. He contributed to the development of Mendel's Accountant as a biologically-realistic computing tool for understanding the dynamics of mutation, selection, and random drift in natural populations.

Wesley H. Brewer

Fluid Physics International

Wesley Brewer is the sole proprietor of Fluid Physics International, a small consultancy specializing in developing numerical simulation software for modeling complex scientific phenomena. His primary research area is in computational hydrodynamics, but has also been working in computational genetics and numerical weather simulations. Since 2005, he has been part of the Mendel's Accountant development team. Dr. Brewer holds a B.S. in engineering science and mechanics from the University of Tennessee, an M.S. in ocean engineering from the Massachusetts Institute of Technology, and a Ph.D. in computational engineering from Mississippi State University. Since 2007, Dr. Brewer spends much of his time teaching computer science in Korea.

John R. Baumgardner

Department of Earth and Environmental Sciences, Ludwig Maximilians University, Theresienstrasse 41, 80333 Munich, Germany.

Dr. Baumgardner has a B.S. in electrical engineering from Texas Tech University, a M.S. in electrical engineering from Princeton University, and a Ph.D. in geophysics and space physics from UCLA. From 1984 to 2004 he served as a staff scientist in the Theoretical Division of Los Alamos National Laboratory engaged in a variety of research projects in computational physics. Beginning in 2004 he has been part of the team which developed Mendel's Accountant, a computer model for investigating research topics in population genetics. He is currently an adjunct staff scientist in the Department of Earth and Environmental Sciences at Ludwig Maximilians University in Munich, Germany.

Chase W. Nelson

Research Scientist

Chase W. Nelson is a biologist and musician currently pursuing a Ph.D. in bioinformatics and molecular evolution. He graduated from Oberlin College in 2010, where he performed honors research on mutation accumulation in Arabidopsis. While at Oberlin, he became an NSF STEM Scholar in Computation and Modeling, and also took part in several research experiences, including an NIH IDeA Networks of Biomedical Research Excellence Fellowship at the University of Wyoming. He subsequently worked under Dr. John C. Sanford at Rainbow Technologies, Inc., where he examined the power of natural selection in digital organisms. His current studies under Dr. Austin L. Hughes focus on developing computational methods to detect natural selection at the nucleotide level. His design of novel tools for next-generation sequence analysis and geographic information systems earned him an NSF GRFP Award in 2013. During the summer of 2013, he also undertook an NSF EAPSI Fellowship to study rice genetics under Dr. Wen-Hsiung Li at Academia Sinica (中央研究院) in Taipei, Taiwan.

Josiah Seaman

Ph.D. student in Computational Biology at Colorado University, Denver, CO.

Josiah Seaman is a student of Bioinformatics. He has a bachelor's in Computer Science. He is currently working as a Ph.D. student in Computational Biology at CU Denver. His specialties are data visualization and sequence analysis. He is the creator of Skittle Genome Visualizer (dnaskittle.com) which is being used to better understand chromosome structure and organization. The downloadable version is freely available at http://sourceforge.net/projects/skittle/

Donald E. Johnson

Ph.D. in Chemistry from Michigan State University as well as Computer & Information Sciences from the University of Minnesota.

Dr. Don Johnson (see video clips from a presentation) has earned Ph.D.s in both Computer & Information Sciences from the University of Minnesota and in Chemistry from Michigan State University. He was a senior research scientist for 10 years in pharmaceutical and medical/scientific instrument fields, served as president and technical expert in an independent computer consulting firm for many years, and taught 20 years in universities in Wisconsin, Minnesota, California, and Europe. He has made ID-Friendly and Intelligent Design Presentations on most continents, including in Russia, China, Australia, New Zealand, England, and Germany. He now owns and operates Science Integrity with Website www.scienceintegrity.org, which has more details on the books (including excerpts, reviews, and endorsements), as well as interviews, speaking tours, on-line videos, and other information.

Michael J. Behe

Department of Biological Sciences, Lehigh University Bethlehem, PA 18015.

Michael J. Behe graduated from Drexel University in Philadelphia, with a Bachelor of Science degree in Chemistry. He did his graduate studies in biochemistry at the University of Pennsylvania and was awarded a Ph.D. for his dissertation research on sickle-cell disease. From 1978-1982 he did postdoctoral work on DNA structure at the National Institutes of Health. From 1982-85 he was Assistant Professor of Chemistry at Queens College in New York City, where he met his wife. In 1985 he moved to Lehigh University where he is currently Professor of Biochemistry. In his career he has authored over 40 technical papers and two books (*Darwin's Black Box: The Biochemical Challenge to Evolution*, and *The Edge of Evolution: The Search for the Limits of Darwinism*). These books argue that living systems at the molecular level are best explained as being the result of deliberate intelligent design. The books have been reviewed by the New York Times, Nature, Philosophy of Science, Christianity Today, and many other periodicals. He and his wife reside near Bethlehem, Pennsylvania, with their nine children.

Jed C. Macosko

School of Mathematics, Wake Forest University.

Jed C. Macosko is an associate professor of biophysics at Wake Forest University. He graduated from MIT with the Merck award for outstanding scholarship and earned a Ph.D. in biophysical chemistry at the University of California, Berkeley in 1999 for his work on the molecular machinery of influenza, HIV and nerve cells. From 2000 to 2002 his research on molecular machines continued as an NIH postdoctoral fellow in the laboratory of Carlos J. Bustamante and then in 2003 and 2004 as an adjunct assistant professor working with David J. Keller at the University of New Mexico. Since 2004 the Macosko lab at Wake Forest has used *in vivo* and *in vitro* microscopy to study how molecular machines move cargo from one part of a cell to another. His team has developed a novel drug discovery platform based on combinatorial libraries of nucleic acid encoded chemicals. His studies on molecular machines and nucleic acids have resulted in over 25 technical papers, book chapters and submitted patents, which have been cited nearly 1000 times and have provided further evidence for design in nature. He and his wife live in Winston-Salem with their five children.

L. Dent

Visiting Professor of Biology, Pepperdine University Malibu, CA 90263.

Laurieanne Dent is a Visiting Professor of Biology at Pepperdine University where she teaches courses in physiology and zoology. In 2008, she completed doctoral studies at Cornell University in Neurobiology and Behavior with a minor in Genetics and Development. Her dissertation research was focused on brainstem neural circuits which process sub-millisecond communication stimuli from electric organ discharges of weakly-electric African mormyrid fish. As an undergraduate at Texas Christian University, she earned a B.S. in Biology and Secondary Teacher Certification in Composite Science, as well, in 1991. After teaching a diversity of science subjects and levels for several years as a secondary educator, she studied for a M.S. in Biology in physiological ecology at Sam Houston State University in Huntsville, Texas.

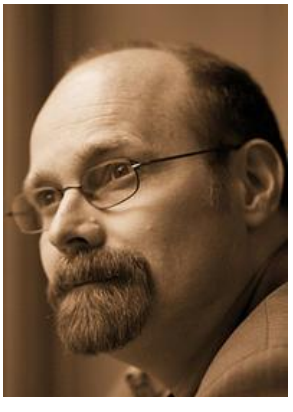
Douglas D. Axe

Director of Biologic Institute Seattle, WA.

Douglas D. Axe is the director of the Biologic Institute. His research uses both experiments and computer simulations to examine the functional and structural constraints on the evolution of proteins and protein systems. After a Caltech Ph.D. he held postdoctoral and research scientist positions at the University of Cambridge, the Cambridge Medical Research Council Centre, and the Babraham Institute in Cambridge. His work has been reviewed in *Nature* and featured in a number of books, magazines and newspaper articles, including *Life's Solution* by Simon Conway Morris, *The Edge of Evolution* by Michael Behe, and *Signature in the Cell* by Stephen Meyer.

Bruce L. Gordon

Associate Professor, Houston Baptist University, USA.



Bruce L. Gordon is associate professor of the history and philosophy of science at Houston Baptist University. He formerly taught science and mathematics at The King's College in New York City, and philosophy at Baylor University, the University of Notre Dame, and Northwestern University. A senior fellow of Discovery Institute's Center for Science and Culture in Seattle, he also served as its research director for a number of years. He holds an A.R.C.T. in piano performance from the Royal Conservatory of Toronto, a B.Sc. in applied mathematics and an M.A. in analytic philosophy from the University of Calgary, an M.A.R. in apologetics and systematic theology from Westminster Theological Seminary in Philadelphia, and a Ph.D. in the history and philosophy of modern physics from Northwestern University in Chicago. The author of a variety of academic articles and the contributing co-editor of two books, he lives in Houston, Texas, with his wife, Mari-Anne

Stuart A. Kauffman

Professor of Biochemistry and Mathematics at the University of Vermont and Professsor of Computational Systems Biology at the Tampere University of Technnology in Finland.

Stuart A. Kauffman is currently Distinguished Professor of Biochemistry and Mathematics at the University of Vermont and Distinguished Professor of Computational Systems Biology at the Tampere University of Technology in Finland. He has also held professorships at the University of Chicago, the University of Pennsylvania, the Santa Fe Institute, the University of New Mexico, the Krasnow Institute at George Mason University, the M.D. Anderson Cancer Center, the University of Calgary, and Harvard Divinity School. A pioneer in the field of complexity theory, he is a biologist, trained as a medical doctor, who studies the origins of life and the origins of molecular organization. Kauffman is the holder of a dozen biotechnology patents and the founder or board member of a number of biotechnology corporations. In 2008 he was elected a Fellow of the Royal Society of Canada. The author of over 180 scientific articles, he is the co-author of one book and the author of four others.

Bruce H. Weber

Emeritus Professor of Biochemistry, California State University at Fullerton, and Robert H. Woodworth Chair in Science and Natural Philosophy Emeritus at Bennington College in Bennington, Vermont.

Bruce H. Weber is Emeritus Professor of Biochemistry, California State University at Fullerton, and Robert H. Woodworth Chair in Science and Natural Philosophy Emeritus at Bennington College in Bennington, Vermont. He is the author of numerous scientific articles and the co-author or co-editor of several books, including *Evolution and Learning* (MIT Press 2003), *Darwinism Evolving: Systems Dynamics and the Genealogy of Natural Selection* (MIT Press 1996), *Evolution at a Crossroads: The New Biology and the New Philosophy of Science* (MIT Press 1989), and *Entropy, Information, and Evolution: New Perspectives on Physical and Biological Evolution* (MIT Press 1988). His research interests are in macromolecular evolution with special emphasis on the application of non-equilibrium thermodynamics to the problems of the emergence of life, and the history of biochemistry, especially the conceptual development of bioenergetics.

Synopsis of Scientific Proceedings: Biological Information - New Perspectives

"This is by far the most rigorous and in-depth re-examination of the sufficiency of neo-Darwinian theory. Never have so many well-credentialed scientists, representing so many disciplines, united so effectively to look beyond the standard mutation-selection paradigm." **- The Editors**

This booklet is a synopsis and limited commentary on the 563 page proceedings of the symposium Biological Information - New Perspectives. The author of this synopsis was the organizer of that symposium and was one of the editors of the proceedings. At this symposium a diverse group of scientists gathered to critically re-examine neo-Darwinian theory, in light of major new evidences that relate to the nature of biological information. This symposium brought together experts in information theory, computer science, numerical simulation, thermodynamics, evolutionary theory, whole organism biology, developmental biology, molecular biology, genetics, physics, biophysics, mathematics, and linguistics.

This synopsis summarizes a milestone book. For over 100 years, it has been very widely believed that the mutation/selection process is sufficient to explain virtually everything within the biological realm. The 29 contributors to this volume bring into serious question this neo-Darwinian paradigm. They use their wide-ranging expertise to carefully examine a series of very fundamental theoretical problems that are emerging. These problems all relate to the exploding field of biological information. Biological information is becoming the primary focus of 21st century biological research. Within each cell there are information systems surpassing the best human information technologies. These systems create what is essentially a biological Internet within each cell. The authors, although holding diverse philosophical perspectives, unanimously agree that the mutation/selection process is not adequate to explain the labyrinth of informational networks that are essential for life.

"For daunted readers of the superb (but very technical and lengthy) "Biological Information - New Perspectives", here comes an extremely condensed version. Editor Sanford offers a splendid synopsis - providing short easy summaries of the book's articles. He adds pointed "significance" paragraphs after each summary – and these leave no doubt about how he interprets the data. Provocative, this synopsis is essential."

- Dr. Bernard Brandstater - Professor of Anesthesiology, Loma Linda University

The complete book, Biological Information – New Perspectives, is available at a greatly discounted price at BINP.org. Alternatively, it can be purchased at either WorldScientific.com or Amazon.com.