

"A bag of powerful intuitions, and a handful of notions"  
The Positive Case for Design

### Paul Nelson

"Easily the biggest challenge facing the ID community is to develop a fully-fledged theory of biological design. **We don't have such a theory right now, and that's a problem.** Without a theory, it's very hard to know where to direct your research focus. Right now, we've got a **bag of powerful intuitions, and a handful of notions** ... but, as yet, no general theory of biological design."

*Touchstone Magazine* 7/8 (2004), pp. 64

### Richard Dawkins

"Biology is the study of complicated things that give the appearance of having been designed for a purpose."

### The MacGuyver Principle

- ▶ "The simpler the solution to a problem, the more intelligence and ingenuity it requires." (Mark Perakh)
- ▶ **Intelligent** design will result in simple, optimal, minimal, solutions to design problems.
- ▶ **Unintelligent** design will result in complex, sub-optimal, sprawling, redundant, solutions to design problems.

### The Design Argument

Evolution couldn't have produced many of the structures in living cells because ...

these structures possess "**Irreducible Complexity**" (Behe)

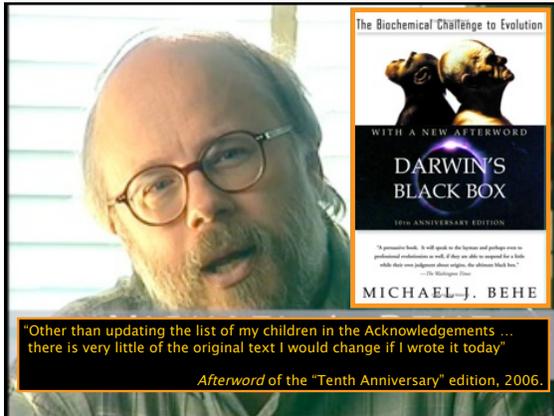
or

these structures possess "**Complex Specified Information**" (Dembski)

"Irreducible complexity is a special case of specified complexity"  
Dembski, *No Free Lunch*, 289

1996

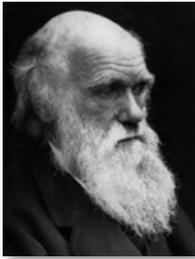
1997



The Biochemical Challenge to Evolution  
WITH A NEW AFTERWORD  
**DARWIN'S BLACK BOX**  
TENTH ANNIVERSARY EDITION  
"A persuasive book. It will speak to the layman and perhaps even to professional evolutionists as well. If they are able to respond for a little while, their own judgment should settle the debate itself."  
—The Washington Post  
**MICHAEL J. BEHE**

"Other than updating the list of my children in the Acknowledgements ... there is very little of the original text I would change if I wrote it today"  
Afterword of the "Tenth Anniversary" edition, 2006.

### Charles Darwin



"If it could be demonstrated that any complex organ existed which could not possibly have been formed by numerous, successive, slight modifications, my theory would absolutely break down."  
"But I can find no such case."

### Michael Behe

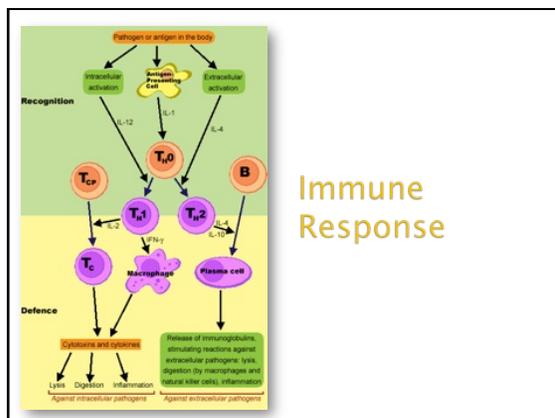
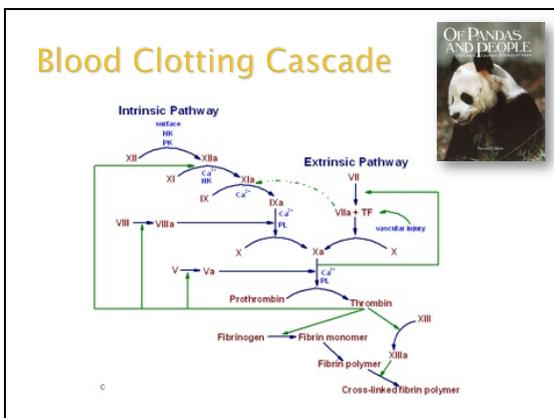


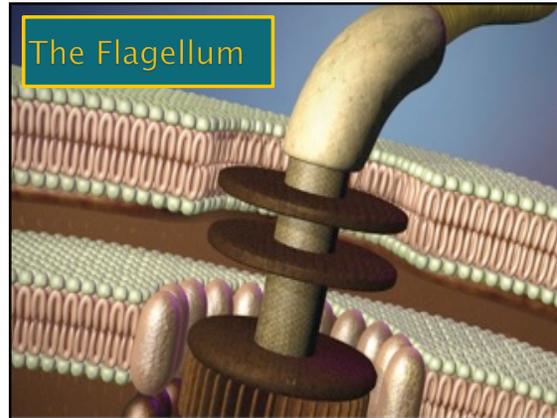
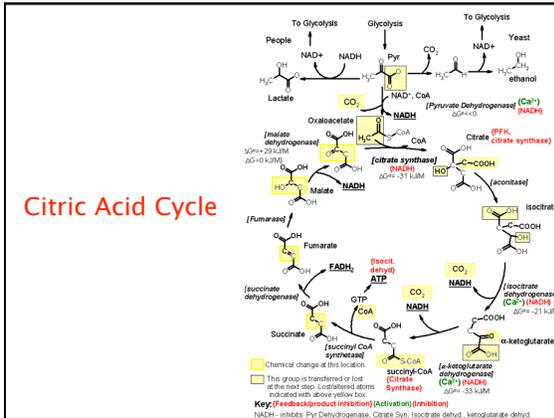
- Claims that Darwinism cannot explain biochemical complexity
- "Irreducibly complex systems ... cannot evolve in a Darwinian fashion"
- "Purposeful arrangement of parts" implies Design

### Behe's Neo-Paleyite Argument

- We infer design whenever parts appear arranged to accomplish a function
- The strength of the inference is quantitative and depends on the evidence; the more parts, and the more intricate and sophisticated the function, the stronger is our conclusion of design
- Aspects of life overpower us with the appearance of design
- Since we have no other convincing explanation for that strong appearance of design ... then we are rationally justified in concluding that parts of life were indeed purposely designed by an intelligent agent

2006, p. 265





### A Momentous Breakthrough

"The result ... is a loud, clear, piercing cry of *"design!"* The result is so unambiguous and so significant that it must be ranked as one of the greatest achievements in the history of science. The discovery rivals those of Newton & Einstein, Lavoisier & Schrödinger, Pasteur & Darwin. The observation of the intelligent design of life is as momentous as the observation that the earth goes round the sun or that disease is caused by bacteria or that radiation is emitted in quanta" (233)

But result is ignored due to implications.

### Michael "Call me Ishmael" Behe

"[I]f random evolution is true, there must have been a large number of transitional forms between the Mesonychid and the ancient whale. Where are they?"

1994

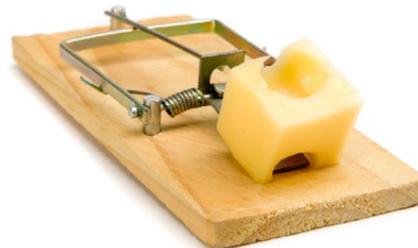
### Irreducible Complexity

"By irreducibly complex I mean a single system composed of several well-matched, interacting parts that contribute to the basic function, wherein the removal of any one of the parts causes the system to effectively cease functioning. ... An irreducibly complex biological system, if there is such a thing, would be a powerful challenge to Darwinian evolution." (39)

## Testing Behe is Difficult

- ▶ He acknowledges the driving forces of evolutionary change
  - natural selection, genetic drift, founder effects, gene flow, meiotic drive, gene duplication, transposition ...
- ▶ "The production of some biological improvements by mutation and natural selection – by evolution – is quite compatible with intelligent design theory."
  - i.e. design exists only when and where evolution cannot explain it

## A mouse trap



## A biological trap



## Variation



**Drosophyllum:**  
Passive glue trap, no motion

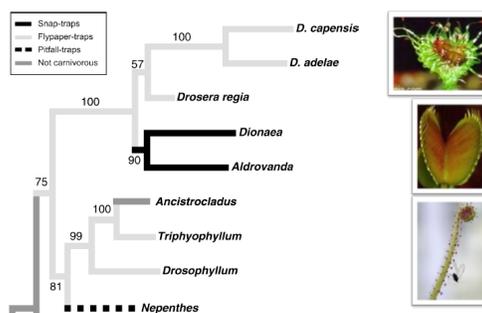
**Sundew (Drosera rotundifolia):**  
Active glue trap, slow motion

## Insectivorous Plants (1875)



"The[se] six known genera...all capture insects. This is effected by *Drosophyllum* ... solely by the viscid fluid secreted from their glands; by *Drosera*, through the same means, together with the movements of the tentacles; by *Dionaea* ... through the closing of the blades of the leaf. In [this] last genera rapid movement makes up for the loss of viscid secretion. ... The parent form of *Dionaea* ... seems to have been closely allied to *Drosera*, and to have had rounded leaves, supported on distinct footstalks, and furnished with tentacles all round the circumference, with other tentacles and sessile glands on the upper surface."

## Molecular Evidence Vindicates Darwin



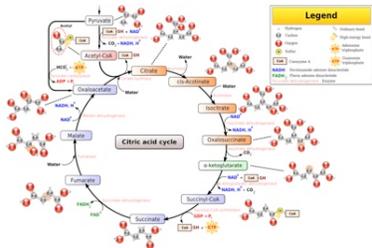
## What About "Odd" Design?



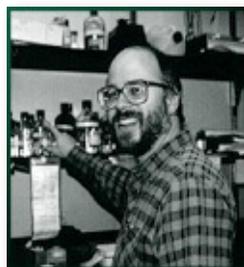
"Features that strike us as odd in a design might have been placed there by the designer for a reason – for artistic reasons, for variety, to show off, for some as-yet-undetected practical purpose, or for some unguessable [sic] reason."



## Evolution of Biochemical Systems



## Behe claims that ...



"[T]here is no publication in the scientific literature – in prestigious journals, specialty journals, or books – that describes how the molecular evolution of any real, complex, biochemical system either did occur or even might have occurred." (p. 185)

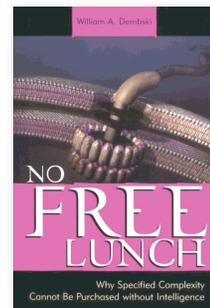
## ID's "clear and daring prediction"



"Darwinists **will not begin** filling in **plausible, testable** scenarios for any of the irreducibly complex cellular systems."

Thomas Woodward, *Darwin Strikes Back: Defending the Science of Intelligent Design* (2006), p. 78

## The Flagellum



## The Flagellum and ID

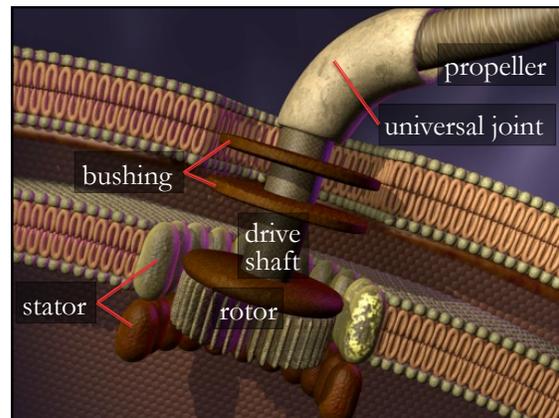
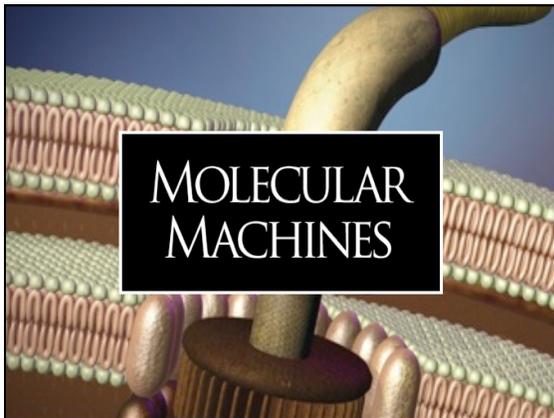


The flagellum "has a machinelike *irreducible complexity*, which is an empirical marker of design because it rules out step-by-step evolution through selection. Take one part away from the flagellum and its rotary system won't work ... Its forty parts, all of them precisely shaped proteins, are *prima facie* evidence for an intelligence behind life, and the flagellum is just the tip of the iceberg. The cell is chock-full of such complex, multipart systems that continue to defy a step-by-step Darwinian explanation."

Woodward, 2006, p. 11

## Behe's Logic

1. **Observation:** The cell contains biochemical machines in which the loss of a single component may abolish function.
2. **Assertion:** Any of these machines that are missing a part is, by definition, non-functional and leaves natural selection with nothing to select for.
3. **Conclusion:** These machines could not have been produced by natural selection.



## Indispensable Bacterial Motor Parts

- 30\* different protein parts ... for the motor's **structure**
- 10\* more protein parts ... for sensor and control **circuitry**
- 10\* more protein parts ... to **construct** the motor



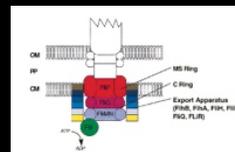
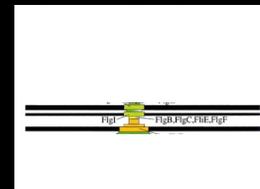
\*These numbers may vary in different species of bacteria



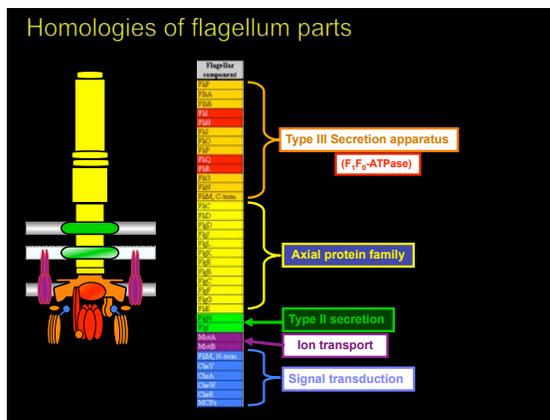
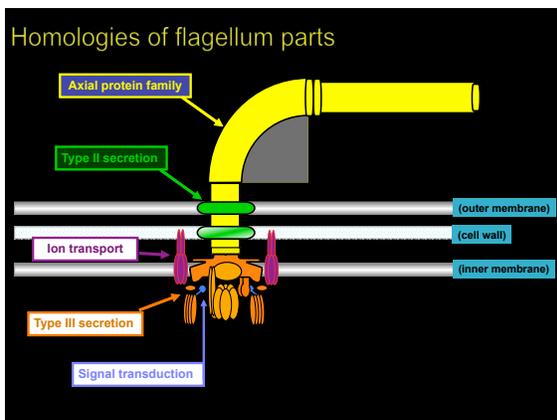
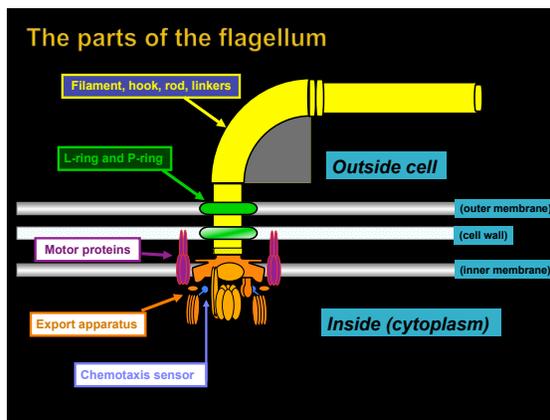
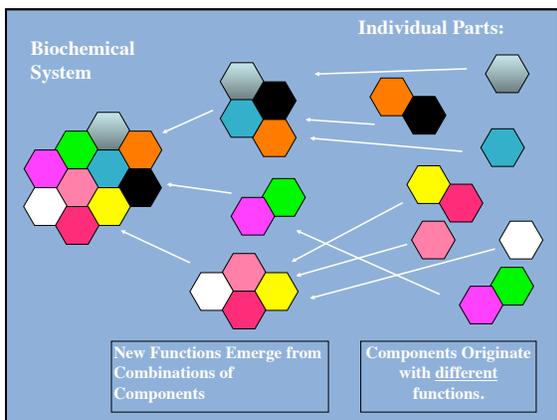
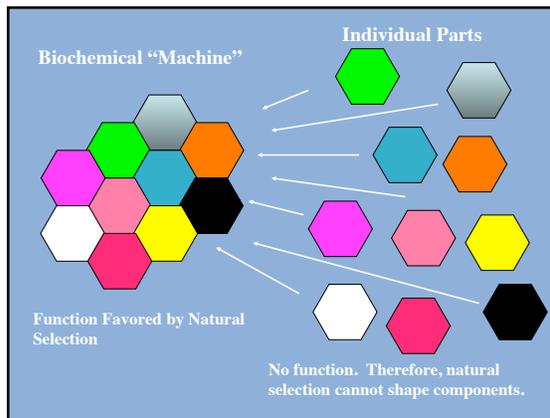
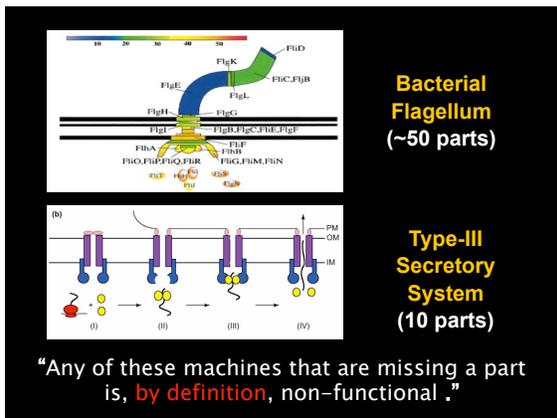
THE ARGUMENTS FOR AND AGAINST NEO-DARWINISM

EXPLORE EVOLUTION

Start with the 50-part bacterial flagellum. . . . And let's take away 40 of the parts:



Leaving just 10. What's left should be non-functional according to Behe.

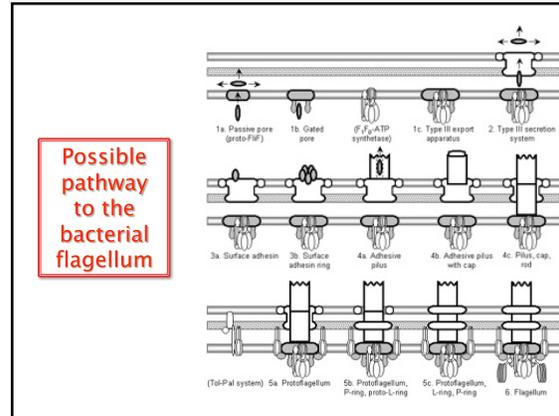


Number of peer-reviewed journal articles:

17

Out of these, the number that Behe has ever written about:

0



Stage	Function of core system	Analogs
1. Primitive type III export system and precursors	Export	Passive inner membrane pores Gated pores Export systems (e.g. sec system)
2. Primitive type III secretion system	Secretion	Secretion systems (Table 4, Figure 5)
3. Surface adhesin	Adhesion	Outer membrane adhesins (Table 4)
4. Type III pilus		Pili (Table 4, Figure 5b,c)
5. Protoflagellum	Dispersal	Random dispersal mechanisms (Vogel, 1994) Dispersal by modern flagella (Dusenberry, 1997)
6. Flagellum	Motility	Motility systems (Table 3, Figure 5d)

### THE ORIGIN OF NEW GENES: GLIMPSES FROM THE YOUNG AND OLD

Table 1. Molecular mechanisms for creating new gene structures

Mechanism	Process	Examples	Comments	References
Exon shuffling		Acetylcholinesterase, pigweed, Pdx1	~10% of exons in eukaryotic genes have been formed by exon shuffling	8, 32, 40, 62, 65-68, 100
Gene duplication		CGP, CM, PNAE1B	Many duplications have probably subfunctionalized	9, 11, 20, 36, 39, 47, 48, 100
Non-gene duplication		PGAM2, Pkg2, PACH1, PACH2, Spire1	1% of human DNA is not devoted to new genomic locations	23, 43, 61, 76, 80-82, 107-110
Mobile element		HLA-DP-1, human DAF, Arginase mRNA, mDCC1 mRNA	Generated 4% of new exons in human protein-coding genes	16, 78, 111, 112
Exon-intron shuffling		acetylcholinesterase (yeast), Bacteriophage phi11, multi/antiMUS	Most often reported in prokaryotes and newly reported in eukaryotes	18, 20, 113
Gene fusion		Fatty acid synthase (yeast), Klu10E, Sbc	Involved in the formation of ~25% of prokaryotic genes	21, 22, 42, 114, 115
De novo origin		ATP1A, BCL110A, GDC0090A	Rare for whole gene origin but, signs of the route for partial gene origin	52-63, 116, 117

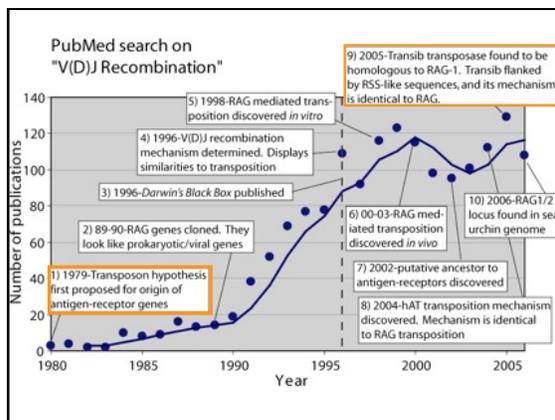
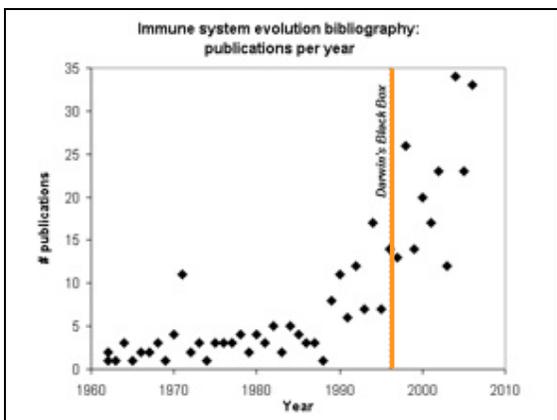
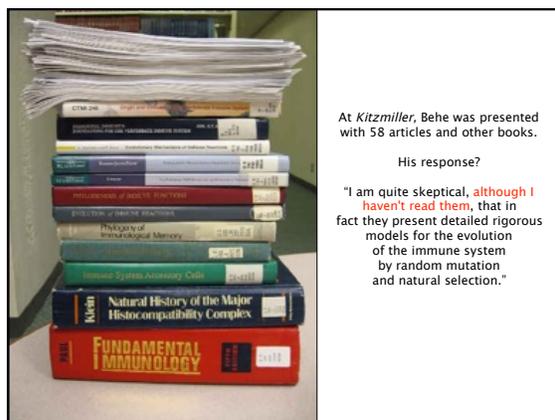
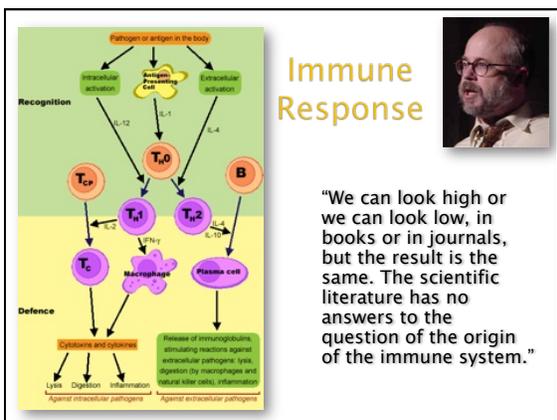
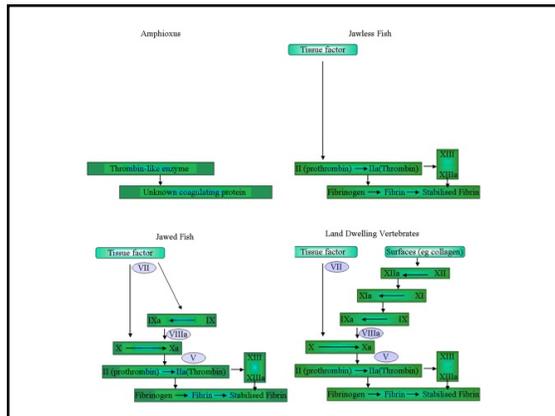
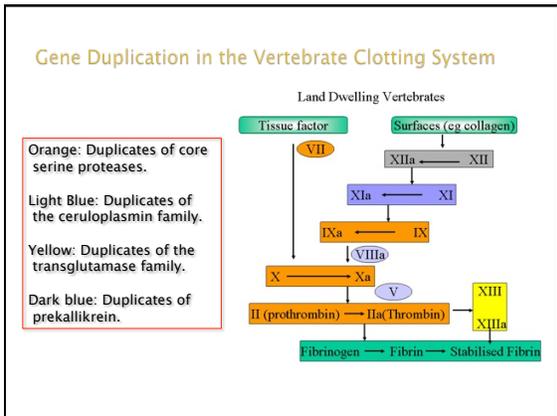
ATP1A, acetylcholinesterase; CGP, chaperone; PNAE1B, 1-pyrenyl-2-iodoacetamide; DAF, deoxy-acylating factor; HLA-DP-1, major histocompatibility complex (MHC) class II; PACH1, protein arginase domain 1; PACH2, protein arginase domain 2; PGM2L, polyoma non-coding long-term RNA; PNAE1B, non-coding RNA; Sbc, sperm-specific DNA strandbreakase chain; UET, tumor susceptibility gene.

### Importance of Duplication

Creation of new copy allows evolution of new function while maintaining old function.

### Mechanism by which an ancestral trypsinogen gene was transformed into an Antifreeze GlycoProtein gene

Chen et al. (1997) "Evolution of antifreeze glycoprotein gene from a trypsinogen gene in Antarctic nototheniid fish" PNAS 94: 3811.



### Leave the research to someone else

- Q. And I'm correct when I asked you, you would need to see a step-by-step description of how the immune system, vertebrate immune system developed?
- A. Not only would I need a **step-by-step, mutation by mutation analysis**, I would also want to see relevant information such as what is the **population size of the organism** in which these mutations are occurring, what is the **selective value** for the mutation, are there any **detrimental effects** of the mutation, and many other such questions.
- Q. And you haven't undertaken to try and figure out those?
- A. I am not confident that the immune system arose through Darwinian processes, and so I do not think that such a study would be fruitful.
- Q. It would be a waste of time?
- A. It would not be fruitful.

### William Dembski



"Behe's challenge was not simply to find a Darwinian explanation for the origin of a biochemical machine, but to find a *detailed* Darwinian explanation for the origin of an *irreducibly complex* biochemical machine."

No Free Lunch, 2002, p. 269.

### A Double Standard



"It's not ID's task to match your pathetic level of detail in telling mechanistic stories."

### Eric Rothschild



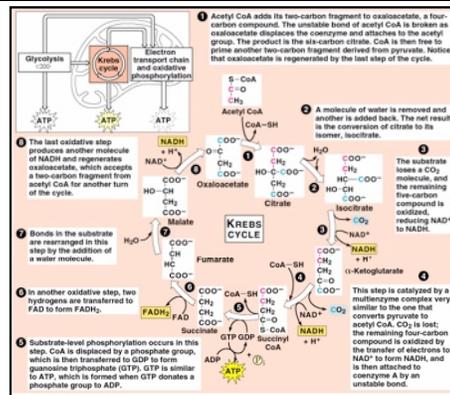
"Thankfully, there are scientists who do search for answers to the question of the origin of the immune system. It's the immune system. It's our defense against debilitating and fatal diseases. The scientists who wrote those books and articles toil in obscurity, without book royalties or speaking engagements. Their efforts help us combat and cure serious medical conditions. By contrast, Professor Behe and the entire intelligent design movement are doing nothing to advance scientific or medical knowledge and are telling future generations of scientists, don't bother."

### Judge John Jones, III



"[Behe] was presented with fifty-eight peer-reviewed publications, nine books, and several immunology textbook chapters about the evolution of the immune system; however, he simply insisted that this was still not sufficient evidence of evolution, and that it was not "good enough" ..."

We find that such evidence demonstrates that the ID argument is dependent upon setting a scientifically unreasonable burden of proof for the theory of evolution."



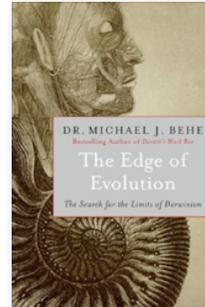


## Michael Behe

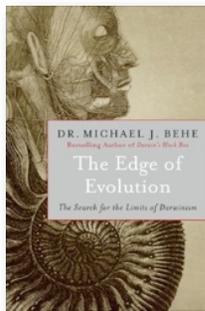


"There is no publication in the scientific literature – in prestigious journals, specialty journals, or books – that describes how the molecular evolution of any real, complex, biochemical system either did occur or even might have occurred."

(p. 185)

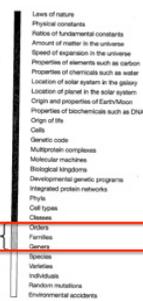


## The Edge of Incoherence



*The Surprising Depth of Fine Tuning of Nature for Life on Earth*

*The Tentative Edge of Random Evolution*



## "Fine Tuned" Arthropoda

- Domain
    - Kingdom
      - Phylum
        - Class
          - Order
            - Family
              - Genus
                - Species
- ▶ Subphylum Chelicerata
    - Class Merostomata (horseshoe crabs, eurypterids)
    - Class Pycnogonida (sea spiders)
    - Class Arachnida (spiders, ticks, mites)
  - ▶ Subphylum Crustacea
    - Class Remipedia
    - Class Cephalocarida
    - Class Branchiopoda (fairy shrimp, water fleas, etc.)
    - Class Maxillopoda (ostracods, copepods, barnacles)
    - Class Malacostraca (isopods, amphipods, krill, crabs, shrimp, etc.)
  - ▶ Subphylum Uniramia
    - Class Chilopoda (centipedes)
    - Class Diplopoda (millipedes)
    - Class Insecta

## Accepts common ancestry



1. **Vitamin C pseudogene:** "Both humans and chimps have a broken copy of a gene that in other mammals helps make vitamin C." (71); "It's hard to imagine how there could be stronger evidence for common ancestry of chimps and humans." (72)
2. **Hemoglobin pseudogene:** "[C]ompelling evidence for the shared ancestry of humans and other primates comes from ... a broken hemoglobin gene." (71)

- ▶ At the origin of life: "intelligent design is quite compatible with the view that the universe operates by unbroken natural law, with the design of life perhaps packed into its initial set-up." [166]
- ▶ Three billion years later: "Explicit design appears to reach into biology to a certain level, to the level of the vertebrate class, but not necessarily further" [220]

## “Front loading” as a Mechanism

“Suppose that nearly four billion years ago the designer made the first cell, already containing all of the irreducibly complex biochemical systems discussed here and many others. (One can postulate that the designs for systems that were to be used later, such as blood clotting, were present but not ‘turned on.’...)” (DBB 227–8)

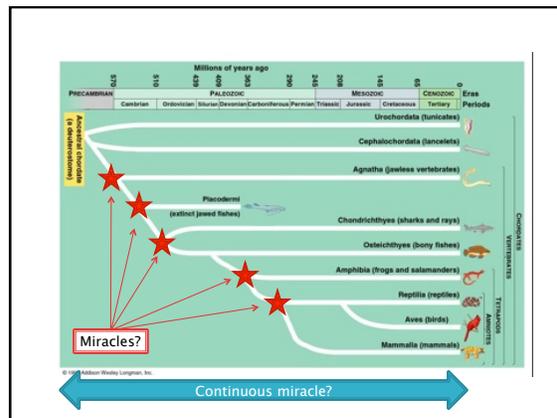
The diagram illustrates five types of chromosomal mutations: Deletion (removal of a segment), Duplication (repetition of a segment), Inversion (reversal of a segment), Insertion (addition of a segment), and Translocation (exchange of segments between non-homologous chromosomes).

**Behe himself admits ...**

“[G]enes that are useless in the real world are not rewarded; the genes are rapidly lost or degraded by mutation.”

## Vertebrate Classes

- Domain
  - Kingdom
  - Phylum
  - Class
  - Order
  - Family
  - Genus
  - Species
- › Hyperoartia (Lampreys)
  - › Chondrichthyes (Cartilaginous fish)
  - › Actinopterygii (Ray-finned fish)
  - › Sarcopterygii (Lobe-finned fish)
  - › Amphibia (Amphibians)
  - › Sauropsida (Reptiles and Birds)
  - › Mammalia (Mammals)



## Non-random mutation

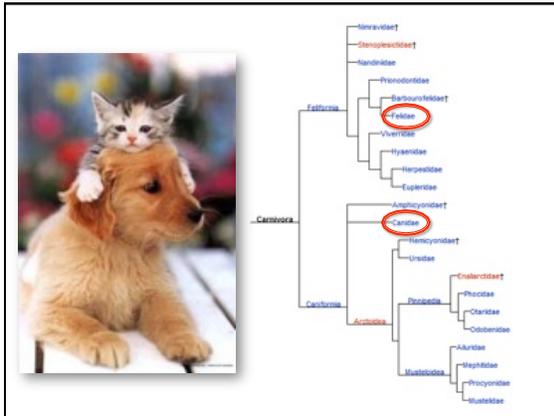
- › “Most mutations that built the great structures of life must have been nonrandom.” [82]
- › “Random mutation does not account for the ‘mind-boggling’ systems discovered in the cell. So what does? If random mutation is inadequate, then (since common descent with modification strongly appears to be true) of course the answer must be nonrandom mutation.” [165]

## Cats, Dogs & Elephants

**Medved:** “What you’re talking about really is the leaps, aren’t you. I mean the kind of random mutations, or allegedly random mutations, who [*sic*] create a new species.”

**Behe:** “Yeah, well I wouldn’t call it species. I’d, I’d go a little higher, maybe genus or something in biology. Biology has a number of levels and you might be able to get, say, from a wolf to a dog using random mutation and natural selection. But I don’t think you can get from a dog to a cat or a precursor organism and get from a dog to a cat or certainly to an elephant.”

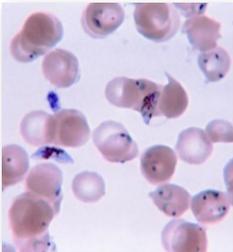
The Michael Medved Show June 5<sup>th</sup> 2007.



### “exquisitely purposeful arrangement of parts”

- ▶ astonishingly complex, coherent systems
- ▶ stupendously complex systems
- ▶ enormously complex cellular mechanisms
- ▶ startlingly complex pathway of flagellum assembly
- ▶ staggering complexity of modern biology
- ▶ tremendously complex elegant complexity
- ▶ stunning complexity
- ▶ enormously complex coherent molecular machinery
- ▶ elegant molecular outboard motors
- ▶ elegant immune system
- ▶ intricate genetic control programs
- ▶ stupendously intricate cellular machines
- ▶ sophisticated living machinery
- ▶ highly sophisticated, automated mechanisms
- ▶ ultrasophisticated molecular machinery

### Plasmodium falciparum



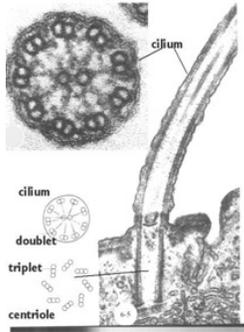
“Here’s something to ponder long and hard: Malaria was **intentionally designed**. The molecular machinery with which the parasite invades red blood cells is an **exquisitely purposeful arrangement of parts.**” (p. 237)

### Natural Dys-Theology

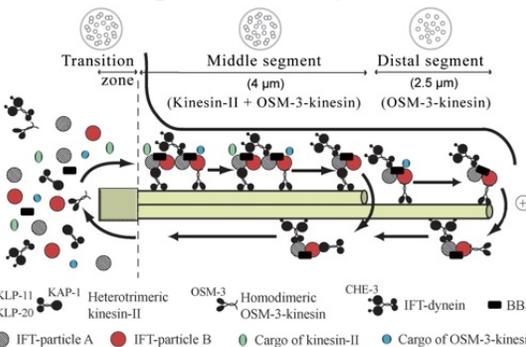


Malaria kills between one and three million people a year, most of them children in Sub-Saharan Africa. It “was **intentionally designed**”.

### The Eukaryotic Cillium



### Intra-Flagellar Transport (IFT)



Transition zone | Middle segment (4 μm) | Distal segment (2.5 μm)

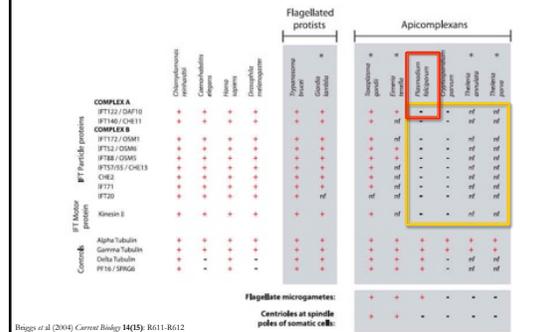
(Kinesin-II + OSM-3-kinesin) | (OSM-3-kinesin)

Legend:  
 KLP-11, KAP-1, Heterotrimeric kinesin-II, OSM-3, Homodimeric OSM-3-kinesin, CHE-3, IFT-dynein, BBS, IFT-particle A, IFT-particle B, Cargo of kinesin-II, Cargo of OSM-3-kinesin

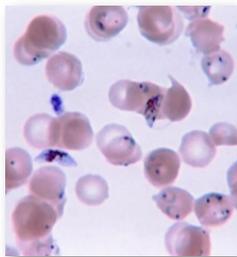
**Behe:**  
 “Irreducible Complexity Squared”

“IFT exponentially increases the difficulty of explaining the irreducibly complex cilium. **It is clear from careful experimental work with all ciliated cells that have been examined, from alga to mice, that a functioning cilium requires a working IFT.** The problem of the origin of the cilium is now intimately connected to the problem of the origin of IFT. [p. 94]

“More than one way to build a cilium”



**Plasmodium falciparum**



**Jerry Coyne**

“If ID were science, we could ... ask Behe to produce a complete step-by-step accounting of what God (sorry, the Intelligent Designer) did when He designed the cilia. And of course Behe would not be able to do that – nor does he even try. IDers never produce their own “scientific” explanation of life. They just carp about evolution. And as evolutionists explain one thing after another, IDers simply ignore these successes and move on to the ever-dwindling set of unsolved problems in which they continue to see the hand of God.”

“The Great Mutator” *The New Republic* 18<sup>th</sup> June 2007

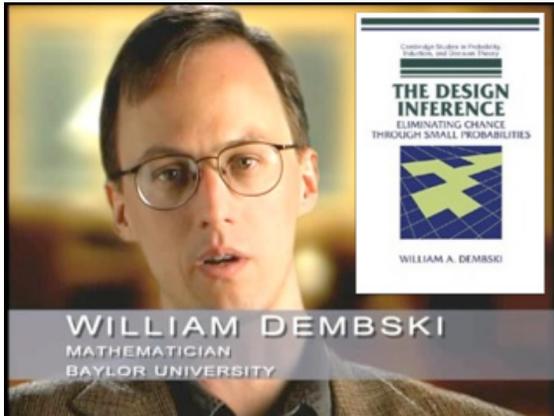
**Jerry Coyne**

“So let us put some empirical questions to Behe, since his theory is supposedly scientific.

- [1] Which features of life were designed, as opposed to evolved?
- [2] How exactly did the mutations responsible for design come about?
- [3] Who was the Designer?
- [4] To what end did the Designer work?

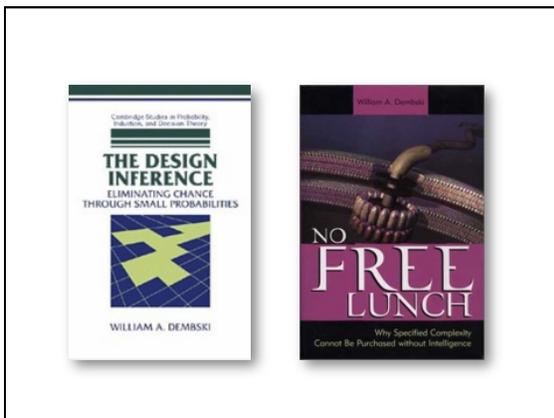
**[4] The Designer’s Goal?**

- ▶ “What we sense, as elaborated through modern science’s instruments and our reasoning, is that we live in a universe fine-tuned for intelligent life.”
- ▶ “Parts were moving into place over geological time for the subsequent, purposeful, planned emergence of intelligent life.”



**Rob Koons**  
Philosopher of Religion, UT Austin

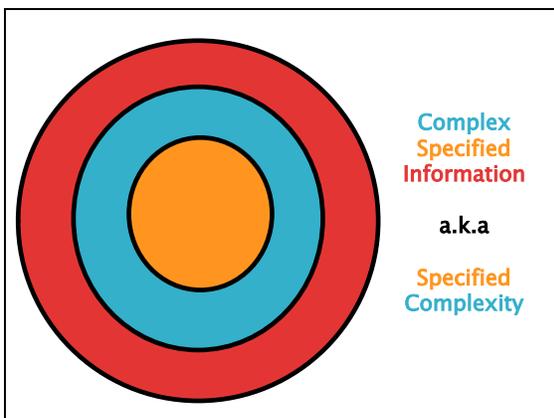
“Dembski is the Isaac Newton of information theory, and since this is the Age of Information, that makes Dembski one of the most important thinkers of our time.”



**The Design Inference**

1.  $Loc(B)$
2.  $(\forall H \in \mathfrak{H}) sp(B, H)$
3.  $(\forall H \in \mathfrak{H}) sp(B, H)$
4.  $(\forall X)(\forall H \in \mathfrak{H}) [Loc(X) \& sp(X, H) \& SP(X, H) \rightarrow \neg ch(X, H)]$
5.  $\neg reg(B)$
6.  $reg(B) \vee (\exists H \in \mathfrak{H}) ch(B, H) \vee det(B)$

Conclusion :  $det(B)$



**Requirements**

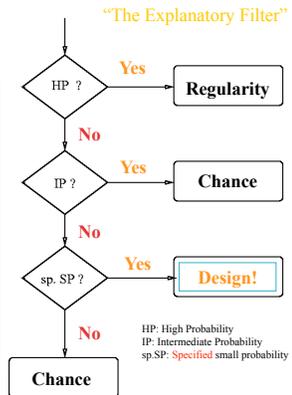
- ▶ **Contingency:** There is a choice (vs. necessity)
- ▶ **Complexity:** Not so simple that the object can be explained by chance
- ▶ **Specification:** Object exhibits a pattern characteristic of intelligence

## Specification



- ▶ "Specification depends on the knowledge of subjects. Is specification therefore subjective? Yes."
- ▶ "Everything depends on what [one] knows, believes, determines, and provisionally accepts"
- ▶ Therefore, specification depends on **current** state of knowledge.
- ▶ **Something is specified if an ID supporter says it is!**
- ▶ This is ultimately a "God of the Gaps" type argument

I can detect design!



## Measuring information

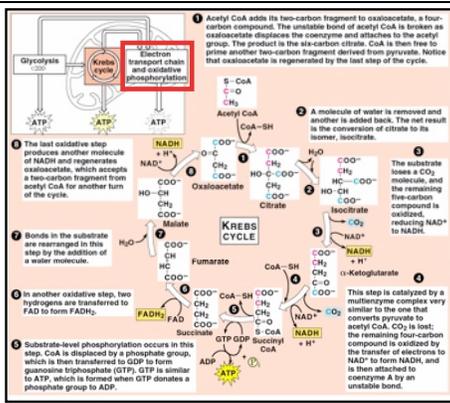
- ▶ Information is seen as a removal of possibilities (decrease in uncertainty)
- ▶ Information (surprisal, entropy) is given by:

$$I_x = -\log_2 p_x$$

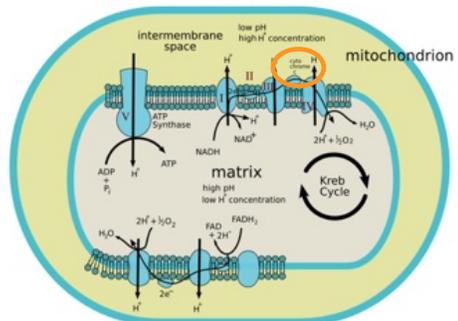
## CSI and the information cut-off



- ▶ CSI is "any specified information whose complexity exceeds 500 bits of information"
- ▶ This is Dembski's *Universal Probability Bound* (UPB) as 500 bits has a probability of  $10^{-150}$ .



## Electron Transport Chain

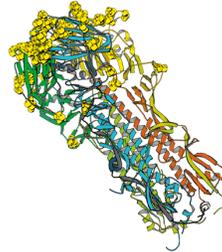


### Cytochrome C



- ▶ Part of the electron transport chain (ATP production)
- ▶ Highly conserved across groups
- ▶ Weighs in at 233 bits, therefore **not** CSI, according to Dembski's criteria
- ▶ *Cyt-c* could have arisen by chance.

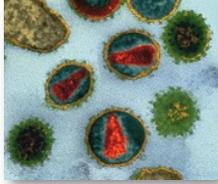
### Protein Binding Sites



- ▶ Human splice acceptor sites contain on average **9.4 bits** of information.
- ▶ Below 500 bit limit, but clearly CSI.



### What about the bad stuff?



- ▶ Viruses, oncogenes, and "jumping genes" cause diseases (e.g. ebola, avian flu, AIDS), cancers and genetic disruption
- ▶ All have information content **beyond** the 500 bit limit of Dembski.
- ▶ According to Dembski they must have been designed.

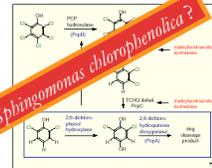
### Evolving a Biochemical Pathway ... in 65 years (or less)

TIBS 25 - JUNE 2000

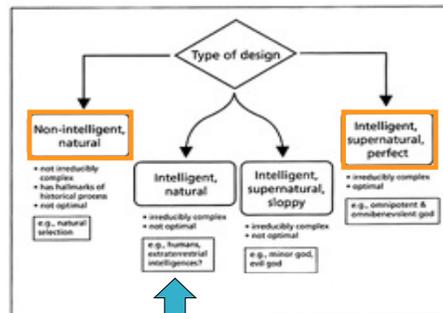
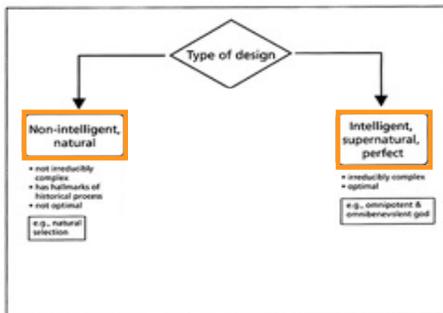
Evolution of a metabolic pathway for degradation of a toxic xenobiotic: the patchwork approach

Shelley D.

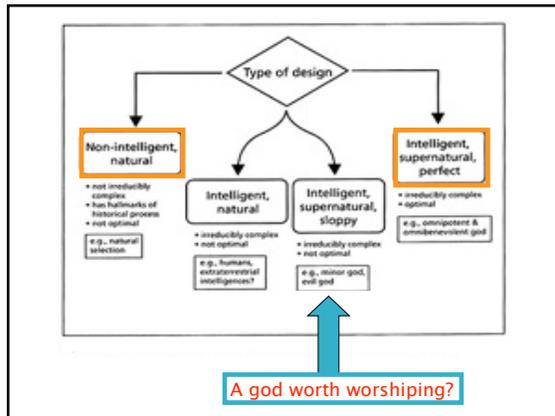
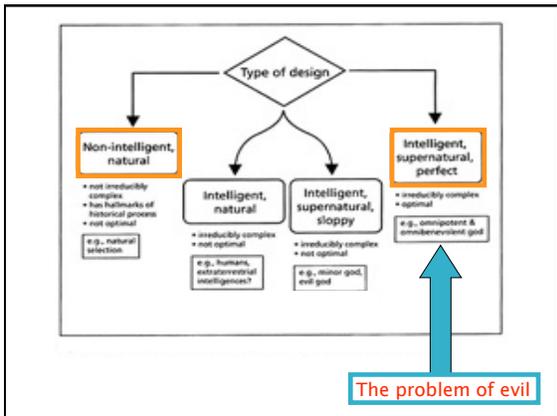
The pathway for degradation of the pesticide pentachlorophenol in *Sphingomonas chlorophenolica* evolved in the past few decades by the recruitment of enzymes from two other catabolic pathways. The first and third enzymes, pentachlorophenol hydrolase and 2,4-dichlorophenol dioxygenase, may have originated from enzymes in the degradation of a naturally occurring chlorinated phenol. The second enzyme, a reductive dehalogenase, may have evolved from a maleylacetoacetate isomerase normally involved in degradation of tryptone. This apparently recently assembled pathway does not function very well: pentachlorophenol hydrolase is quite slow, and tetrachlorohydroquinone dehalogenase is subject to severe substrate inhibition.



Why does the Designer care about *Sphingomonas chlorophenolica*?

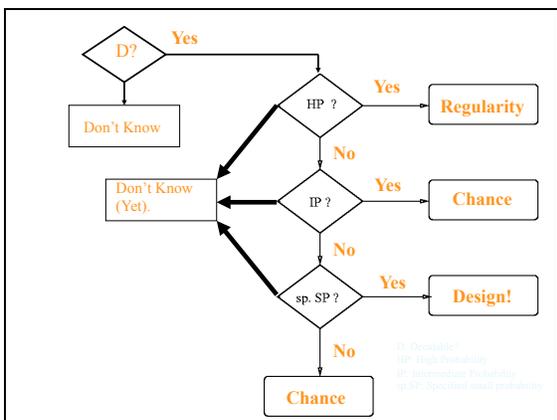
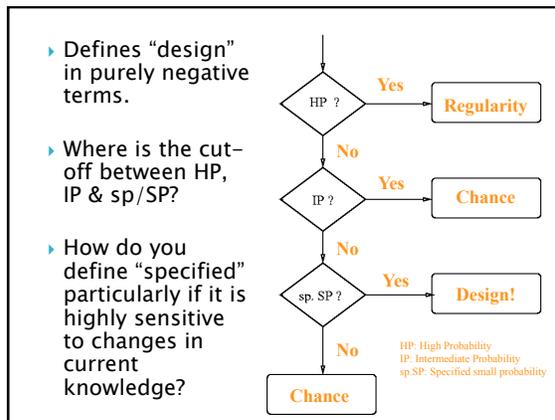


Who designed the agent?



**What is the "unit" of examination?**

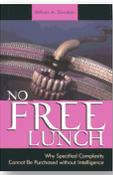
- Section of DNA
- Gene
- Gene family
- Functional group of genes
- Whole genome
- All genomes on Earth
- All genomes in Universe?



**Dembski "Does The Math" (1997)**

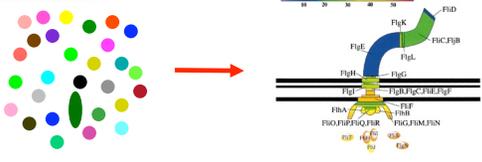
"Do the calculation. Take the numbers seriously. See if the underlying probabilities really are small enough to yield design."

*The Design Inference*, p. 228.



"I show that undirected natural processes like the Darwinian mechanism are incapable of generating the specified complexity that exists in biological organisms."

How? By a calculation showing that the probability of spontaneous assembly of the proteins in the flagellum lies beyond the range of the "universal probability bound" ( $1 \times 10^{-150}$ )



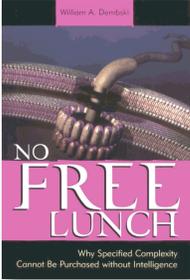
### Dembski 2008



"I've pretty much dispensed with the EF. It suggests that chance, necessity, and design are mutually exclusive. They are not. Straight CSI is clearer as a criterion for design detection."

<http://www.uncommondescent.com/intelligent-design/some-thanks-for-professor-olofsson/#comment-299021>

### Dembski 2008



"The challenge for determining whether a biological structure exhibits CSI is to find one that's simple enough on which the probability calculation can be convincingly performed but complex enough so that it does indeed exhibit CSI. The example in NFL ch. 5 doesn't fit the bill."

<http://www.uncommondescent.com/intelligent-design/some-thanks-for-professor-olofsson/#comment-299021>

2794-2799 *Nucleic Acid Research*, 2000, Vol. 28, No. 14 © 2000 Oxford University Press

### Evolution of biological information

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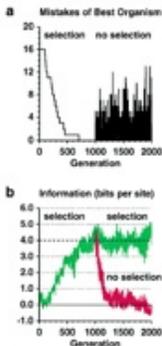
Received March 7, 2000; Revised and Accepted May 25, 2000

**ABSTRACT**

How do genetic systems gain information by evolutionary processes? Answering this question precisely requires a robust, quantitative measure of information. Fortunately, 50 years ago Claude Shannon defined information as a decrease in the uncertainty of a receiver. For molecular systems, uncertainty is closely related to entropy and hence has clear connections to the Second Law of Thermodynamics. These aspects of information theory have allowed the development of a straightforward and practical method of measuring information in genetic control systems. Here this method is used to observe information gain in the binding sites for an artificial protein in a computer simulation of evolution. The simulation begins with zero information and, as in naturally occurring genetic systems, the information measured in the fully evolved binding sites is close to that needed to locate the sites in the genome. The transition is rapid, demonstrating that information gain can occur by punctuated equilibrium.

These measurements show that there is a subtle connection between the pattern of binding sites and the size of the genome and number of sites. Relative to the potential for changes at binding sites, the size of the entire genome is approximately fixed over long periods of time. Even if the genome were to double in length while keeping the number of sites constant,  $R_{genome}$  would only change by 1 bit, so the increase is quite insensitive. Likewise, the number of sites is approximately fixed by the physiological functions that have to be controlled by the recognizer. So  $R_{genome}$  is essentially fixed during long periods of evolution. On the other hand,  $R_{genome}$  can change rapidly and could have any value, as it depends on the details of how the recognizer contacts the nucleic acid binding sites and those numerous small contacts can mutate quickly. So how does  $R_{genome}$  come to equal  $R_{protein}$ ? It must be that  $R_{genome}$  can start from zero and evolve up to  $R_{protein}$ . That is, the necessary information should be able to evolve from scratch.

The purpose of this paper is to demonstrate that  $R_{genome}$  can indeed evolve to match  $R_{protein}$  (12). To simulate the biology, suppose we have a population of organisms each with a given length of DNA. This fixes the genome size, as in the biological situation. Then we need to specify a set of locations that a recognizer protein has to bind to. That fixes the number of sites, again in nature. We need to code the recognizer into



### Schneider's ev program

Using a starting random base sequence of 256 bases, and a population of 64, *ev* generated (using random mutation and natural selection with *no* human intervention) a "CSI" binding site in 704 generations.

<http://www.lecb.ncifcrf.gov/~toms/paper/ev/>

### Efficiency?

- This gain in information required the "death" of 32 organisms in each of 704 generations, i.e. 22,528 deaths.
- This is **very** inefficient, yet a site with a probability of 1 in 500,000,000,000,000,000 (1=68.76 bits) is generated in ~1000 generations
- The **entire** human genome (~4,000,000,000 bits) could evolve in a billion years
- This does **not** consider sexual recombination or realistic population size

## Beating the UPB

- ▶ UPB = No of particles  $\times$  Planck time  $\times$  Age of Universe
- ▶ UPB =  $10^{80} \times 10^{45} \times 10^{25} = 10^{150}$
- ▶ "All the probabilistic resources in the known physical universe cannot conspire to render *remotely probable* an event whose probability is less than this universal probability bound." (Dembski, *The Design Revolution*, p. 87)
- ▶ Schneider looked for the evolution of 512 bits in a *small* (n=512) asexual population of creatures. He allowed *one* mutation per generation.
- ▶ He defeated the UPB in *15,000* generations

Living things create information ("specified complexity") via environmental selection and random mutations. Living things and their environment are the "intelligent designer"

## Major Criticisms with CSI

- ▶ Information theory approach is just probability "dressed-up" as the framework offers nothing new
- ▶ How do you assign the probability thresholds? What is the cut-off between CSI and CI, etc.?
- ▶ The "Explanatory Filter" is a method for *justification of belief* rather than detection

## Reaction to CSI

- ▶ **Organismal Biologists** ... little time for abstractions, especially as no predictions are made
- ▶ **Mathematicians** ... little notice
- ▶ **Philosophers** ... negative due to problems with Explanatory Filter
- ▶ Number of papers using Dembski's method?