

HOW ANTI-EVOLUTIONISTS ABUSE MATHEMATICS

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1. INTRODUCTION

The Reverend William A. Williams was not one of Darwin's bigger fans. In [21] he wrote

The evolution theory, especially as applied to man, likewise is disproved by mathematics. The proof is overwhelming and decisive. Thus God makes the noble science of mathematics bear testimony in favor of the true theories and against the false ones.

Needless to say, this will come as news to most biologists.

The Reverend, writing in 1925, relied heavily on the authority of the Bible in making his arguments. That same year saw biology teacher John Scopes hauled into a Tennessee courtroom, charged with teaching scientific theories that were in conflict with scripture¹ Modern critics of evolution take a more subtle approach, preferring to cloak their dubious religious arguments in the raiment of science. They call themselves Intelligent-Design Theorists (IDT's), the term "creationist" being now somewhat disreputable.

Granville Sewell of the University of Texas at El Paso is one representative of this movement. In [19] he opined, basing himself on Michael Behe [1], "I believe there are two central arguments against Darwinism, and both seem to be more readily appreciated by those in the more mathematical sciences." The two arguments were that natural selection is not capable of building complex organisms, and that Darwinism is in conflict with the second law of thermodynamics. In making these arguments he simply ignored the vast literature addressing both subjects, so as to give the impression that logical fallacies obvious to you or me have somehow eluded our benighted colleagues in the life sciences. It is an arrogance typical of

¹The question of whether Darwinism is genuinely in conflict with the Bible was not addressed at the trial.

the ID movement; armchair philosophers believing they can refute in a day what thousands of scientists have built over the course of a century.

ID theorists offer a wide array of arguments in defense of their position, some of them explicitly mathematical. I will consider some of these arguments here.

2. THE BASIC ARGUMENT FROM IMPROBABILITY

The hemoglobin in our blood is comprised of 574 amino acids arranged in a precise sequence. Any major deviation from this sequence leads to a nonfunctional molecule. We also note that there are twenty sorts of amino acids used by living organisms. Is it plausible that a mechanism based on chance, as Darwinism plainly is, could have produced hemoglobin?

Mathematician David Foster doesn't think so. In [7] he offers the following:

The specificity of hemoglobin is described by the improbability of the specific amino acid sequence occurring by random chance. Such specificity is capable of exact calculation in the permutation formula:

$$P = \frac{N!}{n_1!n_2! \cdots n_{20}!}.$$

... In the case of hemoglobin, and substituting in the above formula the specific numerical value of the solution, $P = 10^{654}$.

Of course, N denotes the total number of amino acids in the sequence, while n_i denotes the number of occurrences of the i -th amino acid.

Hemoglobin is a dummy variable in this argument, any other complex organic molecule or system would have worked just as well. The logic is always the same: the n parts of the complex system are identified as the points of a probability space. This space is then equipped with the uniform distribution. The origin of the system is modeled as the event of choosing the appropriate n -tuple out of this space. If the system is at all complex, the probability of this event will invariably prove to be too small to be worth bothering with. This argument is a mainstay of creationist literature; it has been applied to DNA, the human eye, and the origin of life in [7], [12] and [14] respectively, among many others. I will refer to it as the Basic Argument from Improbability (BAI).

David Foster [7] is confused on many points (one of them being the difference between a permutation and a combination), but the most important error is the portrayal of Darwinism as fundamentally a theory of chance. Darwinism, as described in [10], has three components:

- (1) Organisms produce more offspring than can possibly survive.
- (2) Organisms vary, and these variations are at least partly heritable by their offspring.
- (3) On average, offspring that vary most strongly in directions favored by the environment will survive and propagate. Favorable variations will therefore accumulate in populations.

Part one is a simple empirical fact. Part two is the realm of chance; the genetic variations exhibited by an organism are random with respect to the needs of that organism. But part three is the antithesis of chance. Natural selection is a lawlike process. It is this aspect of Darwinism that gets left out of the BAI.

Foster's argument assumes that evolution proceeds by "single-step selection." But if the preliminary stages of a complex system are preserved by selection, then complexity can be explained as the end result of a step-by-step process. ²

3. IMPROVING THE BAI

Perhaps we could develop a more sophisticated probabilistic model of evolution. For example, Darwinism can be viewed as a Markov chain. The states of the chain are the genotypes³ of the organisms that have existed throughout history; the transition probabilities are the chances of an organism with genotype ϵ_1 leaving offspring with genotype ϵ_2 . Denote by \mathcal{G} the set of all genotypes.

Define a function $\mu : \mathcal{G} \times \mathcal{G} \rightarrow [0, 1]$ which denotes the degree of difference between two genotypes, say ϵ_1 and ϵ_2 . If $\mu = 0$ then $\epsilon_1 = \epsilon_2$. If $\mu = 1$ then ϵ_1 and ϵ_2 share no genes. Let the random variable $\zeta(t)$ represent the state of the system in time t . A central tenet of Darwinism asserts that the relevant genetic variations between parent and child are small

²Popular-level treatments of the power of cumulative selection versus single-step selection, and Darwinian explanations of complexity can be found in the books by Dawkins [4] and [5].

³The genotype of an organism is the sum total of its genes.

relative to the size of the genome, so

$$\text{Prob}\{\zeta(t+1) = \epsilon_j \mid \zeta(t) = \epsilon_k\} \rightarrow 0 \text{ as } \mu(\epsilon_j, \epsilon_k) \rightarrow 1.$$

Let us take $\zeta(0) = \epsilon_0$ as representing the genotype of some ancient organism, one that is simple relative to the complexity we see today. The evolutionary path followed by the descendants of this organism trace out a path through our Markov chain,

$$\zeta(0) \rightarrow \zeta(1) \rightarrow \zeta(2) \rightarrow \dots .$$

Given our present understanding of genetics, we can say that the future states of the random variable ζ are independent of its past states, the hallmark of a Markov process.

We need one more ingredient to transform our Markov chain into a model for Darwinism. Let $f : \mathcal{G} \rightarrow \mathbb{R}$ associate to each genotype its fitness.⁴ Now let ϵ be a genotype containing a system composed of the parts $\rho_1, \rho_2, \dots, \rho_k$; we will write $\epsilon = [\rho_1, \dots, \rho_k]$. The state ϵ is a descendant of the state ϵ_0 which we assume did not contain the ρ_i . For selection to preserve the parts of the system as they appeared, we must have the following:

$$f(\epsilon) > f([\rho_1, \rho_2, \dots, \rho_{k-1}]) > \dots > f([\rho_1]) > f(\epsilon_0).$$

The addition of each part must increase the fitness of the genotype. Further, we can assert that f satisfies some sort of additivity law, since each part of the system can be viewed as increasing the fitness of the system. Say:

$$f(\epsilon) = f(\rho_1) + f(\rho_2) + \dots + f(\rho_k).$$

We can say that a particular state ϵ is accessible to a Darwinian mechanism if there is a path through our chain on which f satisfies the above conditions.

This line of argument is pursued by David Berlinski in [3]. So far it is simply a model within which to model Darwinian explanations of complexity. The alleged refutation of Darwinism arises from the following definition:

Definition 1. *A system $[\rho_1, \rho_2, \dots, \rho_k]$ is **irreducibly complex**, hereafter denoted **IC**, if $f(\epsilon) = 0$ for all $\epsilon \in \mathcal{G}$ such that $\rho_i \in \epsilon$ and $\rho_j \notin \epsilon$ for some $1 \leq i, j \leq n$. If ϵ is a state containing such an irreducibly complex system, then we will say that ϵ is irreducibly complex.*

⁴The fitness of a genotype depends partly on the environment in which that genotype finds itself, but that is ignored for the moment.

Theorem 3.1. *If ϵ is irreducibly complex then it is not accessible to a Darwinian mechanism.*

Do IC systems exist in nature? Well, Berlinski’s definition of IC is a mathematization of a definition given by biochemist Michael Behe in [1]. Behe defined a system as IC if it involves several parts working together to perform some function, such that the removal of any part from the system results in the nonfunctionality of the machine. Examples of such systems are the human blood clotting cascade ⁵, or the flagellae used for locomotion by some bacteria.

Thus by taking ρ_1, \dots, ρ_n to be the various parts of, say, the blood-clotting cascade, we have our example of a system satisfying Berlinski’s definition of IC. It follows that the natural world is replete with systems inaccessible to Darwinian pathways.

It’s an impressive argument, but wrong for at least three reasons.

In [3] Berlinski claims that his definition of IC entails Behe’s, but this is not correct. A system is IC in Behe’s sense if the removal of one part of the system results in the non-functionality of the system. It is IC in Berlinski’s sense if the organism can derive no benefit from possessing only one part of the multipart system. These are plainly not the same. There are at least two sorts of explanation for how the individual pieces of an IC system can benefit an organism, even without the other parts of the system in place:

- (1) They might perform the same function in isolation as they do in the finished system, but not as well. This mode of explanation is used by Miller in [17], in the case of the clotting cascade, and by Dawkins in [5] in the case of the vertebrate eye.
- (2) They might initially have performed a different function but have been later coopted for their present purpose. In [11] paleontologists Stephen Jay Gould and Elisabeth Vrba coined the term “exaptation” for this phenomenon. Two examples are the evolution of the three bones in our inner ear from homologous bones in the reptilian jaw ⁶ as described in [9], and the origin of the Krebs cycle ⁷ as described in [16].

In 1996 Behe [1] made the audacious claim that the technical literature on evolution is silent with regard to the formation of irreducibly complex systems. This charge was shamelessly

⁵The details of the clotting cascade and a detailed discussion of its evolution can be found in [17]. This fine book contains a chapter refuting Behe’s arguments.

⁶There is an extraordinary series of fossils documenting this change.

⁷This refers to the series of chemical reactions that releases energy from food.

repeated by Sewell in 2000 [19] -though Kenneth Miller [17] had meanwhile cited numerous examples from the technical literature to show this to be false.

The point is that Berlinski's definition of IC is far more restrictive than Behe's. Thus, systems that are IC in Behe's sense are known to exist but are not inaccessible to Darwinian mechanisms. Systems that are IC in Berlinski's sense are inaccessible to Darwinian mechanisms, but are not known to exist.

This is the most serious flaw in Berlinski's model, but there are two others worth mentioning. The first is that notions of irreducible complexity treat the parts of a complex system as if they are discrete entities that either exist in their complete, perfected glory, or do not exist at all. This is not realistic. The parts of a complex system become gradually differentiated over the course of many generations. Therefore, asking what happens to a system when one of its parts is summarily removed is a question of little evolutionary importance.

Finally, Berlinski's argument given here is one of a class of arguments based on the proposition that "genotype space" is too vast to be searched effectively by natural selection acting on chance variations. Complex organisms represent islands of functionality in a sea of nonfunctional genotypes, you see. This brings us to the second difficulty with Berlinski's framework. His insistence that the fitness function f be properly increasing on any sequence of adjacent states in a Darwinian pathway ignores the possibility that mutations can be neutral. In other words, we might have $f(\epsilon_j) = f(\epsilon_{j+1})$ for some j . The overwhelming majority of mutations are neutral in this sense. This vastly increases the number of genotypes that are accessible to Darwinian pathways. Two examples of the importance of neutral mutations in molecular evolution are given by [6] and [15].⁸

4. THERMODYNAMICS

Sewell also argued that Darwinism runs afoul of the laws of thermodynamics. Evolution requires a decrease in entropy over time, whereas a cherished principle of physics says that is impossible. Since Sewell recognizes that the second law applies only to closed systems (which the Earth is not), it is unclear what the problem is. His claim that "natural forces

⁸Berlinski presses his argument further by introducing ideas from the theories of finite-state automata and linguistics, but these arguments are no better than the ones considered here

do not cause extremely improbable things to happen” is pure gibberish. Does Sewell invoke supernatural forces to explain the winning numbers in last night’s lottery?

The fact is that natural forces routinely lead to local decreases in entropy. Water freezes into ice and fertilized eggs turn into babies. Plants use sunlight to convert carbon dioxide and water into sugar and oxygen, but Sewell does not invoke divine intervention to explain the process. Certainly the question of how the input of energy into the environment of the early Earth led to the creation of all that we see around us is a fascinating and important one. That explains the large number of scholarly articles published on the subject every year. But thermodynamics offers nothing to dampen our confidence in Darwinism.

5. AN INTRODUCTION TO POPULATION GENETICS

The ability of natural selection to craft complex adaptations out of chance variations is contingent upon two assumptions:

- (1) Beneficial mutations occur with sufficient frequency.
- (2) A beneficial mutation, once it occurs in an individual, will spread through the population.

Biologists have developed mathematical models to aid in addressing these points. The sub-discipline of biology devoted to analyzing such models is called population genetics.

I begin with a very simple model. Our genes are found in long strings, called chromosomes, in the nuclei of our cells. Typically we imagine a chromosome divided into individual regions called loci. The bit of DNA found at a particular locus is referred to as an allele. Let us consider a single locus which, in each individual in the population, contains one of two alleles. Denote these alleles by A_1 and A_2 .⁹ Assume that the species in question reproduces sexually and that the offspring inherit two copies of each gene, one from each parent. Then members of the population will either possess two copies of the A_1 allele, two copies of the A_2 allele, or one copy of each. I will refer to these three cases as genotypes A_1A_1 , A_2A_2 and A_1A_2 , respectively. Let us further assume that the A_1 allele appears with frequency p in the population, and A_2 appears with frequency $q = 1 - p$. We can think of p and q as representing the probability that a randomly chosen allele is A_1 or A_2 respectively.

⁹The following mathematical arguments are drawn from the excellent text by Gillespie [8].

Theorem 5.1. (*Hardy-Weinberg*) Let A_1 , A_2 , p and q be as above, and assume that the population mates randomly with respect to this allele. Then in the next generation the genotypes A_1A_1 , A_1A_2 and A_2A_2 will appear with frequencies p^2 , $2pq$ and q^2 respectively.

Of course, this theorem is elementary. Given the simplicity of the model, it is surprising that the Hardy-Weinberg law has proven invaluable in explaining observed data in wild populations.

Next we try to quantify the effect of selection on the frequencies of the alleles A_1 and A_2 . Imagine that the three possible genotypes appear with the frequencies determined by the Hardy-Weinberg law. Then the extent to which a particular allele is represented in the next generation is proportional to its representation in the current generation and the probability that an individual possessing that allele survives long enough to reproduce. Let us denote the constant of proportionality by $\tilde{\omega}$. This constant is often referred to as the mean fitness of the population.

Denote by ω_{ij} , with $i, j \in [1, 2]$, the probability that an individual of genotype A_iA_j survives to reproduce. If we now let $f(A_iA_j)$ denote the frequency of genotype A_iA_j in the next generation, we find

$$f(A_1A_1) = \frac{p^2\omega_{11}}{\tilde{\omega}}, f(A_1A_2) = \frac{2pq\omega_{12}}{\tilde{\omega}}, f(A_2A_2) = \frac{q^2\omega_{22}}{\tilde{\omega}}.$$

Since the sum of the three frequencies should be 1, set

$$\tilde{\omega} = p^2\omega_{11} + 2pq\omega_{12} + q^2\omega_{22}.$$

Let us denote by p' the frequency of the A_1 allele in the new generation. Then we can say

$$p' = \frac{p^2\omega_{11} + pq\omega_{12}}{\tilde{\omega}}.$$

Note that each A_1A_2 individual possesses only one copy of the A_1 allele).

So what can we say about the change in frequency of the A_1 allele as time passes? One further calculation yields

$$\begin{aligned} \Delta p &= p' - p \\ &= \frac{p^2\omega_{11} + pq\omega_{12} - p\tilde{\omega}}{\tilde{\omega}} \\ &= \frac{pq[p(\omega_{11} - \omega_{12}) + q(\omega_{12} - \omega_{22})]}{p^2\omega_{11} + 2pq\omega_{12} + q^2\omega_{22}}. \end{aligned}$$

The quantity pq is referred to as the genetic variation of the population. It is maximized when $p = q = \frac{1}{2}$.

Suppose now that the A_1 allele confers a selective advantage on the individuals that possess it. Specifically, assume that $\omega_{11} > \omega_{12} > \omega_{22}$. In that case we see that $\Delta p > 0$, indicating that the frequency of A_1 will tend to increase in succeeding generations. By contrast, if A_1 is at a selective disadvantage, so that $\omega_{11} < \omega_{12} < \omega_{22}$, then we have $\Delta p < 0$ and the frequency of A_1 will tend to decrease. This observation can be expressed more succinctly in the equation

$$\Delta p = \frac{pq}{2\bar{\omega}} \left(\frac{d\bar{\omega}}{dp} \right),$$

and in words in the following theorem:

Theorem 5.2. (*Fundamental Theorem of Natural Selection*) *Natural selection always increases the mean fitness of the population, and does so at a rate proportional to the genetic variation.*

A victory for evolution, right? Beneficial mutations will tend to become fixed in the population, and over long periods will accumulate to produce complex adaptations.

Not so fast. Randomness also has a role to play in the change of gene frequencies over time. For example, suppose a single individual in a population has a beneficial mutation. The probability is only one-half that any particular child born to that individual will inherit the mutation. So it is entirely possible that the mutation will be flushed out of the population before it has a chance to spread.

This is one example of a more general phenomenon called genetic drift. Selection is tending to cause beneficial mutations to spread through a population, while drift is tending to remove them. Perhaps a more sophisticated model of population dynamics would have shown that drift is powerful enough to overcome selection, thus effectively falsifying the Darwinian premise of complexity arising from the gradual accretion of small, chance variations.

This line of argument is pursued by physicist Fred Hoyle in [13]. His starting point is the assumption that mutations are far more likely to be harmful than beneficial. How do the handful of beneficial mutations avoid being swamped by the more numerous harmful ones? The answer, known for decades by population geneticists but presented as revelation

by Hoyle, is that the mechanics of sexual reproduction allow beneficial mutations to become “decoupled” from the harmful ones.¹⁰ But sexual reproduction leads to drift, which tends to deplete variation.

Hoyle then points to results like the following:

Theorem 5.3. *Let $Prob_{fix}(p)$ denote the probability that the allele A_1 , appearing with an initial frequency of p , becomes fixed in a population of size N . Then*

$$Prob_{fix}(p) = \frac{1 - e^{-2Nsp}}{1 - e^{-2Ns}},$$

where s denotes the selective advantage conferred by the allele A_1 .

If we assume that A_1 initially appears in a single individual, then we will have $p = \frac{1}{2N}$. Since s is assumed to be small, we can say $e^{-s} \approx 1 - s$. If we then assume that N is large enough so that $e^{-2Ns} \approx 0$, we conclude that $Prob_{fix}(p) \approx s$. So most beneficial mutations are lost without ever having a chance to become fixed in the population. Hoyle concludes from this that it is effectively impossible to string together a large number of beneficial mutations.

Fred Hoyle is no kind of creationist. He doubts neither the truth of evolution nor the existence of a fully naturalistic explanation for it. Indeed, he offers a rather imaginative alternative to Neo-Darwinism based on the premise that the Earth is periodically bombarded with storms of genetic material from outer space. For a brief discussion of why biologists are generally skeptical of this possibility, see [18] and [20].

Hoyle’s argument is wrong for many reasons, the most fundamental being the absurdity of extrapolating to geologic time a mathematical model that is reliable only for short-term data. The dynamics of gene frequencies in wild populations are governed by so many variables that a mathematical model for describing them in the long-term is impossible. For example, the selective value of a particular allele changes with the environment. The population size, and therefore the frequency of a particular allele within it, changes as subpopulations migrate away from the ancestral stock. Animals interact with other animals, which are themselves evolving. Consider also that we have been focusing on one locus, when in reality the selective value of the allele at that locus is certainly affected by the alleles at other loci.

¹⁰A mathematical derivation of this fact can be found in any text on population genetics, [8] being a particularly good one.

There are other problems. Early in his book Hoyle states, "...a considerable fraction of individuals born in every generation exhibit some new mutation, the great majority being harmful in some degree." This premise is entirely false. As indicated earlier, most mutations are neutral. And what of the small probability that a beneficial mutation will become fixed in a population? That only applies to very large populations. Most evolutionists believe that periods of speciation, during which directional evolutionary change accumulates very quickly, occur when small "founder" populations become geographically isolated from the ancestral stock.

This leads us to the most insidious aspect of Hoyle's work. His book offers no index, no bibliography, and only the briefest mention of any other work in population genetics. Most of his book is spent rederiving old results, without giving any indication that they are not original to him. A lay reader will inevitably get the impression that the formidable mathematical machinery employed by Hoyle, coupled with his dismissals of work that came before him, constitutes a devastating attack on Neo-Darwinism. It doesn't.

6. PSEUDOMATHEMATICS

As an academic dispute all this is minor. But it plays in public. ID theorists, much like the creationists before them, know they will not convince scientifically knowledgeable people. Instead, they market their ideas to a public untrained in both the methods and findings of science. And all too often their's is the only viewpoint that is readily available.

When scientists are presented with subjects that invoke the terminology of science to defend nonsense, like astrology and creationism, they use the term *pseudoscience*. I suggest we need a similar term, *pseudomathematics* perhaps, to describe mathematical formalism, used to promote bad arguments. As professional mathematicians, we all have an interest in protecting the integrity of our subject. We have an obligation to be aware of how mathematics is being used in the public square. When we see pseudomathematics, we should not be afraid to identify it.

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