License

Introduction

We first look at some common criticisms of creationism. One classic work is done by Pennock (Pennock 2003) . At the time of publishing, Pennock was at the Lymann Briggs School and Department of Philosophy, Michigan State University according to the publication. The document is more of a manifesto laying out strategies for scientists to defend good science, which is in his work, defined as the theory of evolution.

Criticism 1: Creationists use Argument from Ignorance

Pennock states that creationists use “argument from ignorance”. Argument from ignorance is a classic logical fallacy where a person thinks A is true because of the lack of visible evidence that contradicts A (Walton 2010) . Walton seems to be an expert in this field and has specialised in these sorts of logical fallacies. Though he himself has published literature stating that there are certain cases in which argument from ignorance is non fallacious (Walton 1992) . That however, is out of scope for the time being.

In the context of creationism, one argument which a creationist might fallaciously make is “we cannot know for sure how the world came to be, or that we cannot be sure that evolution has produced the biodiversity that we see today, therefore there must have been a God who made all the biodiversity”. Anecdotally, I may have heard of some who hold to this view. It’s a little cringy, though I hold on to the same axiom at the end of the day. Process is important though, and one ought to take care of how these thought processes arise. Personally, I don’t agree with this line of reasoning either.

I might make the assertion or axiom that things that look “intelligent” should be “intelligently designed” as an axiom. Axiom 2: I would make the observation that physics from quantum mechanics to relativity seem to have a certain mathematical beauty to it. Axiom 3: Mathematical beauty hints at intelligence. If one presupposes these axiomatically, one can arrive at the intelligent design conclusion without the logical fallacy of argument from ignorance. Lennox, an apologist and mathematician might agree with these axioms (Lennox 2009).

Though of course, the axioms themselves are not universally agreed upon. Beauty is subjective and potentially another philosophical can of worms altogether. We shall not discuss it yet.

Okay, we’ll leave it here for now, there are other matters to explore. I’ll probably come back to this later (TBD).

Criticism 2: On Irreducible Complexity

Pennock writes that writers such as Behe developed a concept of irreducible complexity, and how Behe's arguments were refuted when Biologist Allen Orr showed that a Darwinian mechanisms could have indeed produced such systems. The paper is titled Darwin v. Intelligent Design (Again) (Orr 2005)

This was published in the Boston Review, A political and literary forum as originally cited by Pennock, or in Think Autumn in Cambridge University Press. Are these peer reviewed? It seems to be more a forum in the former case or a university magazine rather than a peer reviewed paper.

Now in Orr's writing, he essentially quotes that "evolutionists all know that from the time the earth formed, it took three billion years to evolve the first true cell but only half as long to get human beings from this cell". (Orr 2005) The claims here are quite vague. What is the mechanism and chemistry? Where are the peer reviews? And how did you come up with three billion years? Where are your error bars? And what is the mathematical model used to come up with this timeframe? I need to see how you came up with those numbers. Probability in abiogenesis is as much a big black box if you don't quantify how it happened. Looks like I'll need to search more literature here.

Orr writes that while it is highly unlikely that all the parts of an irreducibly complex system are assembled simultaneously, he asserts that "Behe's colossal mistake" is that no Darwinian solution remains. Orr says that an Behe's colossal mistake is that in rejecting the possibility that irreducibly complex parts can be evolved one part at a time provided the sub parts of the reducibly complex system serves some function initially. He gives the examples that air bladders evolved into lungs. So while lungs are extremely complex in that having one part fail would shut the whole thing down, they could have evolved via Darwinian mechanism. (Orr 2005) Orr then goes on writing that this scenario is not hypothetical and he uses the experience of computer programmers.

Programmers may iteratively develop programs and build lines of code one stage at a time. Each line of code does one job, but as the programmer makes changes, he or she is able to iteratively improve the program until the final piece of software does a more complex job, where all lines of code are needed. (Orr 2005)

I find it amusing however, that he would use a programming analogy, given that program development requires many many steps of intelligent guided design. It is an analogy that serves more to strengthen the argument of intelligent design rather than weaken it. This is because an intelligent programmer or designer is already assumed from the get go in code development.

I'm writing this repository on Github and have experience with various programming languages and with writing and using simulation software. I know how complex things can get. A programmer does NOT randomly program functions and classes which do random things, and assmeble them together by chance. A programmer also cannot do anything without first knowing the syntax of the language. This requires intelligence as well, doesn't it? You don't just randomly type things into the interpreter or compiler and hope it works. And guess what, interpreters and compilers are also intelligently designed by other programmers. These then run on an operating system, such as Windows or Linux. And I know the Linux Kernel is incredibly complex and designed by highly intelligent people. And that's only software, we haven't even discussed design in hardware, as well as the electrical and cooling systems required to support the hardware.

Moreover, a programmer has some end in mind they want to achieve. Whether it is Computational Fluid Dynamics simulation, or mesh building, or building some artificial neural network. Given that end goal, the programmer then breaks down the problem into many parts with the use of Object Oriented Programming to segregate the required functions and data into various classes as is the often the case for C++, Java or C#. A programmer may also choose to organise the required tasks via

Functional Programming, via Haskell perhaps. Regardless of the code paradigm, there will be an intelligent programmer planning how the program runs, and how it should function to meet customer needs. There is more often than not, the presence of well thought out design when it comes to real world software development and research. So, in Orr using the programming analogy, I think it is a very poor choice of analogy in trying to prove his point.

Now, a programming analogy may not carry over directly to biology. But it is worth looking at the underlying scientific work behind the arguments. Orr quotes H. J. Muller's work in 1918 and 1939 "that genes that at first improved function will routinely become essential parts of a pathway. So the gradual evolution of irreducibly complex systems is not only possible, it's expected" (Orr 2005).

On Mutations by Herman J Muller

Now, I'll be interested to go down the rabbit hole of H. J. Muller. This was what I was looking for. Probably come back here and write more. A good place to start is one of Muller's most well quoted papers "The relation of recombination to mutational advance" (Muller 1964) .

Now I'm not quite able to find Muller's original papers. But from a quick internet search, he was awarded a nobel prize for his work in inducing genetic mutations with the use of X-rays (ionising radiation). I'm not a heatlh physics person, but of course, ionising radiation is what I'm meant to know professionally. One possible candidate for the 1938 paper Orr talks about is Muller's work on Radium titled "Report on investigation with Radium". (Muller 1939) I currently am not able to find this report at the time of writing. So I have very little to go on.

Another report is on 1928, where Muller talks about producing mutations with X-rays. (Muller 1928) . Don't know if this is that relevant, but good to read for background anyhow. He basically states that when he blasted X-rays at fruit flies, and then have then mate with non irradiated fruit flies, the offspring of these flies (he refers to them as crosses) have a higher mortality rate than those of the control fruit flies. He refers to the mortalities as "lethal mutations". It's a little frustrating as I don't see a methods section here indicating the rate and dose of exposure. Such information is meant to be bread and butter for any piece of scientific literature. This is not the best of quality due to the lack of materials and methods. But to be fair, hindsight is 20/20, and he published his papers in the 1920s, so we can't be too harsh on him. Nevertheless, you can't really repeat experiments here without those pieces of information.

Presence of such poor quality papers would probably have been higher in those days. But anyhow, let's see his thought process. He has pretty much established

Mutations Require Irreducibly Complex Reproduction Mechanisms to be Expressed

By the way, a thought came to mind, genetic mutation usually happens when genes are passed down from one generation to the next. During these times, the genes of the child are a recombined form of the parent's genes. (I hope I got it right, correct me if I'm wrong). For this to happen, sexual reproduction or asexual reproduction is necessary. For multicelled organisms, the cells must be able to undergo mitosis as well! Without this reproduction, the genes will not be passed down. Even if they are passed within the same generation via viruses or some other means, the genes will not be passed down if the organisms, single celled or otherwise, reproduce. Therefore, the capability to reproduce, sexually or asexually seems quite essential to the process of evolution. This is because even if genetic mutation occurs within an organism when it is alive, the organism will eventually die. If it dies, that genetic information is lost. I must say that for mutation to occur and for those genes to be passed down from generation to generation, the capability for reproduction is a prerequisite. And reproduction, in any of its forms, could be said to be an irreducibly complex mechanism.

And the fact is that, for mutations to be expressed within the cell, the prerequisite is that genes must be expressed. For that to happen, there is also an irreducibly complex process. Today, we see that as there are complex proteins and enzymes which help this process. The transcription factors help to transcribe the information within DNA to mRNA. And the mRNA is meant to go to the ribosome to be translated into a protein. For any of these processes to happen, the cell usually needs some source of energy. Adenosine Triphosphate (ATP). Today, that ATP is produced in the mitochondria. In a supposed prehistoric cell, perhaps the chemicals were different. However, the basic functions of genetic transcription must be there. For that to occur, the cellular machinery and the chemical energy to power that machinery must be present. Where does the energy come from? There must be some other system in place to collect this energy and convert this into energy carrying molecules. All of these systems are strictly necessary for evolution to even start its course for mutation over (supposedly) billions of years.

Now perhaps, I'm mistaken and I need to be corrected somewhere. So I'll just hold this thought for now and read more literature.

back to Muller

In muller's 1928 paper (Muller 1928) , he describes how X-rays cause abberations in fruit flies (Drosophila) and generalises the mutation effect of X-rays to other organisms other than fruit flies based on the works of other researchers. Also, he concludes that radiation from Radium (which we know today as an alpha and beta emitter), can also cause mutation. These are perhaps things we take for granted.

a little about Mendelian Genetics

He also mentioned Mendelian genes. What are meneldian genes? I'll have to look further. Let's start from the review paper, "the genetic basis of Mendelian phenotypes: Discoveries, Challenges and Opportunities" (Chong et al. 2015) . We'll need to get used to some of the jargon used just in the abstract.

1. phenotypes
2. genotypes

Johannsen calls the genotype "sum total of all the genes" in a zygote (or embryo) (Johannsen 1911) . Whereas the phenotype describes the different organisms distinguishable by observable characteristics (Johannsen 1911) . For example, a human is a different phenotype from a bear. From person to person within the same human race, two siblings will have different phenotypes because they have different characteristics which are observable.

Mendelian genetics says that the sum total of all genes of a living organism (genotypes) will cause the different observable characteristics (phenotypes) between organisms. I guess phenotypes and phenomenon have a similar root word, in that they refer to observable facts. So one key idea of Mendelian genetics is that genotype determines phenotype.

Anyhow, Chong et al's paper (Chong et al. 2015) has its focus more about mapping the human genome and using that knowledge to treat disease. It is more focused on clinical advances and understanding "the genetic basis for inherited disease" (Chong et al. 2015) . Not much is said here with regards to origin, and genetic disease is another rabbit hole I cannot explore for now.

More about Mendel, he describes laws of inheritance. One of these laws is the law of random assortment (Davey Smith and Ebrahim 2003) . Mendel's original paper is called experiments in plant hybridisation (Mendel 1996) . This was the paper I was looking for when I wanted to understand more about Mendel's laws and experiments.

So, Mendel had this paper about plant hybridisation. The particular paper I'm reading pertains to a translated version (Mendel 1996) from German, since Gregor Mendel was indeed German speaking. Several corrections have been made since the first translation in 1901 by Baeston (Mendel 1996) . I will assume the translations are reasonably accurate and reliable.

It is noteworthy that Mendel did his studies on plants when doing "plant hybridisation" experiments, or breeding of plant types. Mendel highlights in his methods section that experiments were carefully done to exclude extraneous (irrelevant) circumstances such as unintended cross pollination from other plant species. Mendel also notes that cross breeding does inevitably result in some plants being sterile which can frustrate experiments. However, he notes that "all members of the series developed in each successive generations should be, without exception, subjected to observation". (Mendel 1996)

The language is difficult to say the least, but my working understanding is that the plants are all subject to observation regardless of the sterility or reduced fertility.

Mendel notes in his experiment that "only the most vigorous plants were chosen for his fertilisation experiments" as the weaker plants "afford uncertain results (Mendel 1996) . By that he means that many of the "weakly" plants fail to flower or produce inferior seeds (Mendel 1996) .

Mendel uses a large number of plants in his experiments to deduce the statistical ratio of 3:1. 253 hybrids and 7324 seeds produced a phenotype of 5474 round or roundish seeds, 1850 angular or wrinkled seeds. (ratio 2.96:1) 258 plants yielded 8023 seeds of which 6022 were yellow, 2001 were green. (ratio of 3.01:1). This is quite impressive detective work, it is very thorough and statistically significant. (Mendel 1996)

From these statistics, he comes up with a mathematical model of sorts to describe the propagation of these recessive and dominant genes and their resultant phenotypes. (Mendel 1996) The mathematical model rests on the assumption that the way reproductive cells recombine to form a new zygote (seed) is quite probabilistic.

But yes, reading Mendel's experiments of peas is a little dry. But it is a fundamentally useful model to describe inheritance, and quite impressive for someone in the 1800s to write. It seems he has done his homework quite well. I'll probably continue another time.

Notes resuming 25 feb 2024 punctuational evolution

Now, I'm back to writing. To reiterate, there is this notion that there is irreducible complexity, therefore Darwinian evolution cannot explain it. But that claim was contentious. One paper on this topic is that discussing evolutionary "bursts" and gradual evolution. The evolutionary bursts are known as punctuational bursts (Venditti and Pagel 2008). There is acknowledgement of these "punctuational bursts" of evolution in the paper (Venditti and Pagel 2008).

The fuller theories trying to explain this punctuational bursts are better described in "Punctuated Equilibrium" and "Punctuated Evolution" (O'Brien et al. 2023). O Brien goes through in detail the problems of a squaring the fossil record with gradual evolution in part two of his paper (O'Brien et al. 2023).

O Brien writes that to reconcile the missing fossil records with Darwinian Evolution, one had to either assume the fossil record was incomplete, or else found a new theoretical alternative, Punctuated Equilibrium (abbreviated PE in the paper) (O'Brien et al. 2023). Punctuated Equilibrium postulates that evolution be split into two parts: macroevolution and microevolution. Microevolution more closely resembles Darwinian evolution whereas macroevolution is meant to explain large changes in species in short times. Punctuated Equilibrium postulates that rather than microevolution being responsible for different species, macroevolution is more responsible (O'Brien et al. 2023). In

Punctuated Equilibrium, Eldredge and Gould postulated that macroevolution is more than repeated rounds of microevolution (O'Brien et al. 2023).

What then is macroevolution? Can we observe it like microevolution? And what is its mechanism?

That's it for today, will have to do a more in depth look into macroevolution next time.

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