The Existence of God: An Application of the Poisson Distribution

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Abstract: In his theory of evolution by natural selection, Charles Darwin provided a plausible alternative to Christianity's creation account of human origins. In response, the Christian botanist Asa Gray suggested to Darwin that the variation that drives evolution might be generated by God. Darwin rejected Gray's hypothesis, invoking philosophical naturalism, a hallmark scientific paradigm. Darwin's conclusion was reached on ideological grounds rather than empirical ones. Biological evidence that emerged subsequent to Darwin's time yields a different conclusion. A means to assess the question of the source of genetic variation is provided by fitting the Poisson distribution to counts of mutation and chromosome crossover events at the DNA sites where they occur. A general failure of fit between observational data and the Poisson distribution confirms an exception to the naturalistic paradigm, and thereby provides epistemic access to the existence of God.

Keywords: God; evolution; mutation; recombination; genetics; molecular biology; naturalism; Poisson distribution

Charles Darwin hypothesized that life evolves by natural selection operating on abundant naturally occurring variation. Much discussion has ensued about whether life evolves by natural selection, the effect of differential reproduction rates due to differential fitness. Underlying the discussion is the question of the existence of God, whose creative role is subsumed by the paired processes of random variation and natural selection. The random component commonly associated with natural selection supports atheism. Randomness denies purpose.

However, the question of whether life evolves by natural selection obfuscates the question of God's existence. It is the absence of purpose in the introduction of variation, not the natural selection process itself, that refutes the existence of God. Rather than use natural selection as a proxy test for the existence of God, it is instructive to instead focus on the related, but more fundamental question of whether variation really is random. By rephrasing the question appropriately, it can be seen that evolution in fact points to God. For the purpose of determining whether God exists, natural selection, *per se*, is a red herring. The ultimate creative processes in evolution, genetic mutation and recombination, are demonstrably nonrandom. Moreover, these processes exhibit bias by gene function, revealing purposefulness in the assignment of attributes to individual living beings. This result refutes the scientific premise that all observable phenomena are explicable in natural terms. The nonrandom processes of spontaneous genetic mutation and recombination, unlike any other natural phenomena, provide means to know of the existence of God by observing the world.

Darwin, lacking an understanding of genetics, could not explain the source of the variation on which natural selection acts.[1] Over 150 years after publication of his *On the Origin of Species*,[2] modern genetics attributes genetic novelty to spontaneous DNA mutation and recombination.[3] Mutation accounts for the origin of new alleles, or gene variations.

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Recombination is the source of new combinations of gene alleles, another kind of genetic novelty. Mate choice is a form of recombination, as is the Mendelian mechanism of independent assortment, the shuffling of homologous chromosomes during sexual reproduction. Another form of recombination is the "crossing over" process that shuffles DNA between individual chromosomes during meiosis. Broadly, whereas mutation creates the attributes, recombination creates the individuals that are combinations of these attributes. Ultimately, mutation is the original source of variation, but subsequent recombination supplies significant variation as well.[4]

Asa Gray, an American botanist and contemporary of Darwin, and the primary supporter of his theory in the United States, suggested that God might be the source of the variation on which natural selection acts. Referring to Darwin's inability to account for the source of variation, historian of science Edward Larson explains,

> "Asa Gray ... immediately seized on this gap in Darwin's argument to propose that God guided the evolutionary process by causing the beneficial variations that selection acts upon in evolving new species. Over the years, he developed this insight into a fully articulated theory of theistic evolution, but Darwin rejected it."[5]

Although Asa Gray's ideas were consistent with the empirical observations that Darwin made in his *Origin of Species*, Darwin relied on exclusively natural explanations. In so doing, Darwin conformed to, and helped to establish, the naturalistic paradigm of modern science. Larson explains, "Darwin's theory dispensed with the need for a Creator to design species: Natural processes alone could produce each feature, trait, and instinct of every species."[6]

This naturalistic account implies a random source of variation (Darwin used the word "chance"). Curtis Johnson has identified three key observations about the role of chance in Darwin's thought:

(1) Darwin discovered "chance" as a basic factor in evolution from an early time in his career, perhaps mid-1837.

(2) Darwin understood some important implications of this discovery from a nearly equal early period for how his views would be received, specifically: (1) that "chance" (in its primary meaning for Darwin) would be regarded as a "dangerous" idea (in this he was correct); (2) that he probably had to readjust his own religious views in light of his discovery; (3) that he could not in good conscience pretend to himself or the world that he did not really mean it; (4) that to ensure scientific acceptance of his discovery he would need to cast the role of chance in ways that, while preserving its central meaning, would either obscure its role in his theory or at least make it seem innocuous to otherwise friendly natural philosophers and scientists; and (5) that to accomplish this end he would need to rework his wording in his published writings.

(3) Changes made by Darwin in how he chose to present "chance" in his theory may be of greater significance than any others in the Darwinian corpus. At a minimum they are extremely important in seeing how he "evolved" in mode of expression.[7]

Johnson observes that one way in which Darwin reworked his wording was to adopt the phrase "spontaneous variation" to substitute for "chance variation" in later editions of his *Origin of Species*.[8] Use of the term "spontaneous" persists to this day in describing the occurrence of

genetic mutation. The idea of chance, or random, variation would be developed further in the Neo-Darwinian evolutionary synthesis that later reconciled evolution with Mendelian genetics.[9]

Where Darwin invoked chance, Gray invoked purpose. Based on the facts available to Darwin and Gray, the natural and supernatural explanations were equally valid in terms of their ability to account for variation. In philosophy of science terminology, Darwin's theory of natural selection was underdetermined by the data available at the time. Gray's supernatural explanation was as valid as Darwin's naturalistic one in terms of its ability to explain Darwin's observations about evolution. The tie breaker in this debate was the application of the naturalistic paradigm of science which says that all natural phenomena are explicable in terms of other natural phenomena. Darwin, who had attended a seminary on the intention of entering ministry before his journey to the Galapagos, ultimately adopted the agnostic perspective of his protégé T.H. Huxley. A Gray biography says of Huxley, "His agnosticism, not Gray's argument from design, became the official policy of the Darwinian movement."[10]

The problem with supernatural explanations in science, it has been said, comes down to the need for testability. A publication of the National Academy of Sciences (NAS), explains:

> In science, explanations must be based on naturally occurring phenomena. Natural causes are, in principle, reproducible and therefore can be checked independently by others. If explanations are based on purported forces that are outside of nature, scientists have no way of either confirming or disproving those explanations. Any scientific explanation has to be *testable* -- there must be possible observational consequences that could support the idea *but also ones that could refute it*. Unless a proposed explanation is framed in a way that some observational evidence could potentially count against it, that explanation cannot be subjected to scientific testing.[11]

This, then, gives criteria for scientific truth: reproducibility and testability. Implicit in this statement is the assumption that nonnatural causes cannot be reproduced. In other words, they do not recur in a manner that can be tested. However, a statistical randomness test applied to DNA mutation and crossing over provides a way for the NAS criteria to be met for phenomena whose cause may in fact lie outside of nature. The phenomena are both reproducible and testable. The only factor preventing such a test from establishing divine action is an ideological commitment to naturalism in scientific explanations.

A lesson for the preeminence of naturalism in science can be found in Thomas Kuhn's *The Structure of Scientific Revolutions*.[12] Kuhn's groundbreaking thesis held that, in practice, scientists work within an established discipline. Such a discipline provides examples for the way to do science based on a tried and true framework that has been successful in the past. "Normal science," what practitioners of a particular discipline do day in and day out, consists of "puzzle solving." The current paradigm provides a framework that is accepted as valid and scientific practice involves extending that paradigm to cover new applications. Like puzzle solving, there is an expectation that there will be a way to fit the pieces of data together in a sensible manner. There is presumed to be an answer that is consistent with the established paradigm. The scientist's challenge in interpreting new experimental data is to find how the data fit the prevailing paradigm.

Kuhn showed that scientists can be misled when they appeal to a prevailing explanatory paradigm in spite of evidence to the contrary. His primary example was the Ptolemaic geocentric solar system model that was eventually shown to be wrong and ultimately abandoned

in favor of the Copernican heliocentric system. Is it possible that science has been lulled into blind reliance on the naturalistic paradigm such that it is unable to recognize a miracle if it were to occur? Cornelius Hunter warns that this is exactly what has happened:

> Imagine a scientist who begins to study a nonnatural phenomenon. She is unaware that the phenomenon is not natural, and since today's science seeks only naturalistic explanations, she confines her research accordingly. Perhaps her naturalistic explanations, though not true in this case, can nonetheless somewhat accurately describe the phenomenon and make some useful predictions. In this case naturalism works just fine.
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> But what if not? What if ... the naturalistic explanations are forever stymied -- stymied because they use natural laws and processes to describe a phenomenon that does not follow such laws and processes? By searching and searching, the scientist may find a partial fit. So she may have some success, but there are always unexplained observables -- data anomalies for which the naturalistic explanation cannot account. In this case naturalistic explanations will always be problematic. More data will be collected, further analysis will be done, and theories will be modified or replaced altogether. All good scientific research and -- in our hypothetical example of a nonnatural phenomenon -- wrong.
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> The problem with science is not that the naturalistic approach might occasionally be inadequate. The problem is that science would never know any better. This is science's blind spot. When problems are encountered, theological naturalism assumes that the correct naturalistic solution has not yet been found. Nonnatural phenomena will be interpreted as natural, regardless of how implausible the story becomes. Science has no mechanism to detect the possibility of nonnatural phenomena. It does not consider the likelihood that a phenomenon might not be purely naturalistic.[13]

Ironically, science has explained so much of the natural world that it has never been better equipped than it is now to certify a genuine miracle. A miracle by definition is a violation of the natural order. The domain of science is the natural world. At the same time that science has cemented the naturalistic paradigm by example after example where it has explained natural phenomena in entirely naturalistic terms, it has blinded itself to supernatural phenomena by making naturalism a matter of ideology. Philosophical naturalism, or scientific materialism, refers to this ideological commitment to naturalism in scientific explanations.

In Darwin's day, Asa Gray's suggestion that God supplies the variation for natural selection to act on could not be evaluated empirically because the mechanism for representing genetic information was unknown. Gregor Mendel's work was not even known to Darwin. Even beyond the insights of classical genetics, the molecular basis of genetics would need to be worked out before scientists could begin to assess the processes involved in generating genetic variation. Mendelian genetics was not enough. What would be required is knowledge of the biochemical basis of heredity and the mechanisms of reproduction, the province of molecular biology.

Thomas Hunt Morgan's fruit fly lab was instrumental in reconciling Mendelian genetics with evolutionary theory.[14] Morgan's lab found experimental support for the chromosome theory of inheritance. That theory held that chromosomes are the bearers of genetic information. Historian of science Larson observed, "Thomas Hunt Morgan sealed the bond between Mendel's

laws of heredity and material chromosomes during the early 1910s. In doing so, Morgan's team laid the groundwork for the modern synthesis of genetics and Darwinism that has dominated biological thought ever since."[15]

Morgan's lab found experimental evidence that genes occupy particular locations on the chromosomes. After a long search which included attempts to generate mutations artificially, Morgan identified the "white eye" mutation in male fruit flies (Drosophila), an exception to the normal red eye. This mutation was found to be sex-linked and ultimately enabled the identification of gender as a genetically determined trait, specifically by the presence of certain chromosomes. Discoveries of many more gene alleles followed in subsequent years. These mutations were discrete changes to the nominal values found in most individuals. Changes of the type observed by Morgan's group might serve as the raw material for natural selection when they confer a competitive advantage. The group also identified modifier genes that could explain continuously variable heritable "quantitative" traits such as height.

The mutations identified by Morgan's group could each be identified with a particular location on one of the four Drosophila chromosomes. The observable attributes associated with the mutations were discrete variations from the more common "wild type" alleles, to which Morgan's group gave descriptive names such as "scute", "vermillion", "eosin", "truncate", "bar eye", and "speck". As such, they were similar to the factors originally identified by Mendel in peas -- attributes such as "wrinkled versus smooth", "green versus yellow", and "tall versus short". Some factors are linked, and therefore inherited together, while others are independent. The ones inherited independently are located on different chromosomes while those inherited together are located on the same chromosomes as each other.

Whereas Mendel's factors all assorted independently, some of the fruit fly factors exhibit more complex inheritance patterns. Linked genes occasionally assort independently from the other genes on the same chromosome. These genes "cross over" from one chromosome to the homologous chromosome (the one from the other parent among each chromosome pair). Crossovers allow linked genes to assort independently to some extent even though they lie on the same chromosome as each other. Such crossovers, which are recombinations of existing alleles, constitute a second source of genetic novelty, in addition to mutation. Therein lies the foundational importance of the Morgan group's Drosophila work for evolutionary theory. Morgan biographer Garland Allen summarizes, "The new combinations of old characters, as well as the appearance of additional mutants and their successive recombination, provided the raw material on which natural selection could act."[16]

Morgan hypothesized that the frequency of crossovers between two linked genes would increase with the distance between them on the chromosome, due to the greater number of chances for crossovers to occur provided by the additional chromosomal material. Those that lie close together would seldom cross over while those farther apart would cross over more often. The rate of crossovers between any two genes would correspond to the number of points between the genes at which a crossover could potentially occur. Based on this insight, Alfred Sturtevant, a student in Morgan's lab, constructed the first gene map based on recombination rates between known gene alleles in Drosophila.[17] However, Garland Allen relates that "Sturtevant noted ... that the mapping procedure rested on the assumption that chromosome breaks were equally likely to occur at any point along the length of the chromosome."[18] Sturtevant surmised that differences in map distances could be due to weak spots in the chromosomes rather than differences in the space between chromosome breaks.[19]

Morgan was an experimentalist. In his time, experimentalism was a new approach to science. Morgan criticized the approach of the naturalists as speculative. Experiments allowed testing theories. Hypotheses are affirmed or contradicted by experimental evidence. Experimentalism provides a way to get at the truth by designing experiments to test theories. The approach went beyond simple empiricism. Morgan looked for a way to verify his conclusions independently by a different method when checking observations against a theory.[20] As an example, in the process of checking the gene map based on rates of recombination, he cited cytological evidence that crossovers follow a non-random pattern with respect to chromosome location, an effect observed in Drosophila by Theodosius Dobzhansky.[21] Consequently, the physical distances computed using recombination rates are not accurate, due to unequal rates of crossing over in different genetic regions. Morgan explains:

> An important reservation must be made here -- one that geneticists have always been aware of. We have assumed that the chance of crossing over is the same at every level of the chromosomes. As will be shown presently, this may be inexact. The point is illustrated by a railroad time-table. The time a train takes between stations is a fair measure of their distance apart, but it is not exact. There may be grades or variations in speed, or waits at certain points in consequence of which the time between stations is not always an exact measure of their distance from each other. So it may be with the map distances. For, if crossing over should be more frequent in certain regions than in others, the map distances are only approximately true.[22]

The observation of crossover bias by chromosome location has been confirmed to be the case for all eukaryotic organisms that have been analyzed for such. DNA exhibits hot spots and cold spots of recombination activity in fruit flies, yeast, and mammals. Some DNA sites show high rates of crossovers; others show low rates or no crossover activity.[23] However, there is nothing about the structure of the DNA that would suggest the existence of weak spots to account for the varying rates of crossing over.

Morgan was a militant atheist.[24] His colleague Theodosius Dobzhansky explains,

> …the direction of his scientific activity and his personality are incomprehensible without appreciating Morgan's deep-seated and uncompromising opposition to religion. It can be gleaned from some of his writings, although for obvious reasons he did not talk about it explicitly, except with a few intimates. The main goal of basic biology, in fact of natural science, was to show the invalidity of religious views of man and the universe. To do so one must dispel mysteries enveloping man and the world, because mysteries are the foundations and supports of religion. Because heredity was one of the mysteries, genetics was an important science demystifying this particular phenomenon of nature. Evolution was, needless to say, tremendously important, because it did away with the biblical story of creation of the world and man.[25]

Morgan's ideological commitment to atheism precluded supernatural explanations for physical phenomena. Morgan was fully committed to the naturalistic paradigm of science. The discrepancy between genetic distances and physical distances that he observed, along with the observation that genes occupy particular locations on the chromosome suggest an obvious explanation that Morgan could not see. The observed recombination bias by chromosome

location amounts to evidence of purpose in the assignment of traits to individual beings. Given that genes correspond to particular chromosome loci, the bias in rates of chromosome crossovers by locus amounts to bias for particular gene alleles in the assignment of traits to individuals. Genes correspond to physical traits – their purpose so to say – as the white eye allele illustrates. Bias for crossover events at particular chromosome loci amounts to bias for particular traits. Absent a physical explanation for such bias, the process is inherently teleological. Absent any natural explanation for recombination bias by gene location, the correlation between gene location and gene function means that recombination bias amounts to evidence of divine action. The crossover events exhibit a preference for some functions over others, the very behavior one would expect if genetic attributes were being assigned to individual living beings in a purposeful manner. The process is evidently purposeful.

Morgan's work in classical genetics preceded the revolution in molecular biology that would later reveal the chemical basis of heredity in the form of DNA. Morgan himself did not know that DNA was the carrier of genetic information, nor did he know of the genetic code that specifies the production of proteins of varying lengths by corresponding variable length sequences of DNA containing the genetic code. There were many unanswered questions. Given the success of the experimental approach to biology pioneered by Morgan and his colleagues at the fly lab, the prospect of explaining the mutation and recombination processes in natural terms must have seemed inevitable. Indeed, Morgan commented,

> If the causal factors of variations that are inherited were known, it might be quite unnecessary to consider ultra-naturalistic arguments that attempt to give an "explanation" of evolution. But it cannot be said that the causal factors of such variations have been discovered. ... But with every advance in our knowledge of the chemistry and physics of living material, the possibility of finding a naturalistic explanation seems improved.[26]

Molecular biology was born when James Watson and Francis Crick solved the puzzle of the molecular basis of heredity by elucidating the structure of DNA. The four DNA bases always pair in the same manner, Adenine with Thymine ("AT"), and Glutamine with Cytosine ("GC"). The pairs form the rungs of a spiral staircase-like structure. Since the bases always occur in the same pair-wise combinations, a means for replication is suggested where the rungs are split between each pair and two copies are constructed by completing each new pair by adding the corresponding missing base. Crick and Watson reported their result in 1953.[27] Watson presented the findings to a seminar at Cold Spring Harbor Laboratory attended by Seymour Benzer.[28]

The structure of DNA establishes an expectation for the distribution of point mutations, a term coined by Morgan to signify those mutations caused by a change at a single site on the chromosome, or more precisely now, DNA. Point mutations include base insertions, deletions and substitutions, and are the most common types of human DNA mutation.[29] In DNA, the individual base pairs are naturally isolated from each other. There are no chemical bonds between adjacent bases. Instead, bases are bonded to the sugar-phosphate backbone, which only indirectly holds bases next to each other. The nucleotide backbone carrier is the same regardless of the particular base bonded to it at a given site. The backbone, a sugar-phosphate polymer, is therefore independent of the sequence of bases bonded to it. The two base pair types, GC and AT, are nearly identical to each other in shape, differing by a single additional hydrogen bond in GC pairs.[30] The independence of particular sites from each other is key to the suitability of the

molecule as a stable information storage medium. Support for the independence of the particular base pairs at each site comes from Crick and Watson:

> It should further be emphasized that whatever pair of bases occurs at one particular point in the DNA structure, no restriction is imposed on the neighboring pairs, and any *sequence* of pairs can occur. This is because all the bases are flat, and since they are stacked roughly one above another like a pile of pennies, it makes no difference which pair is neighbor to which.[31]

Considerations such as these suggest that each site is equally likely to mutate. Indeed, Watson's molecular biology text makes the commonly invoked assumption that point mutations, which are rare in practice, are expected to be independent of each other and equally probable at each site on the DNA strand.[32] The expected distribution therefore implies a test to determine whether mutations are random. The Poisson probability distribution provides a means to assess the randomness of point mutations. A goodness of fit test comparing actual observed point mutation frequencies at each site with those predicted by the Poisson distribution constitutes a randomness test.[33] The null hypothesis for the test is that point mutations are randomly distributed versus the alternative that they are not. Given that DNA sites correspond to particular genes, and therefore particular biological functions, the null hypothesis corresponds to the naturalistic explanation and the alternative to the supernatural one. This test meets the NAS criterion that scientific phenomena be testable. The epistemic obstacle to detecting supernatural agency cited by NAS is overcome through its relation as the logical negation of random naturalistic agency. The supernatural alternative hypothesis entails the existence of God.

By way of comparison, a well-known example where the Poisson distribution was used to detect purposeful agency was a study of the pattern of buzz bomb attacks on London during World War II. The British had accurate data on the geographical coordinates of each hit and wanted to know if the bombs were being directed precisely to each target or were simply landing haphazardly. They divided a section of London into small equally sized squares and counted the number of hits in each square. They compared the actual number of hits in each square to the number predicted by the Poisson distribution. In their case, the fit was considered to be a good one. They concluded that the pattern was random and the enemy did not have a highly accurate targeting capability for their guided bombs.[34] The squares making up the London neighborhood in the British study can be compared to the sites on the DNA. The agent directing the bombs in the London study was found to be random.

When the range of explanations for scientific phenomena is expanded to include nonnatural causes, a test for the randomness of spontaneous mutation with respect to DNA site is effectively a test for the existence of God due to the identification of gene function, and therefore purpose, with DNA location. Mutation bias is purposeful since there is no purely natural explanation that would cause it. Confirmation of a random distribution supports the null hypothesis, whereas a nonrandom distribution favors the alternative. The alternatives exhaust all possibilities. Such techniques are part and parcel of the tool kit used by empirical science every day. The hypothesis that mutation is random and the alternative hypothesis that it is not form the kind of null/alternative hypothesis pair that is the standard formulation in empirical science and statistical hypothesis testing in general. Observational data that refute a null hypothesis that mutation is random argue for acceptance of the alternative hypothesis that it is not. A

nonrandom distribution of mutation events is a testable observational consequence of the existence of God, thereby meeting the testability requirement of the NAS.

Seymour Benzer first noted a lack of fit as reflected in hot spots and cold spots of mutation activity on his histogram plots of point mutations of the bacteriophage rII gene. Hot spots and cold spots violate the equal probability of point mutations at each site required by the Poisson distribution. Benzer published his results in 1961 in the Proceedings of the National Academy of Sciences,[35] the scientific organization whose publication identified the testability criterion for natural explanations quoted earlier.

Benzer and Watson had both done genetic research with bacteriophages (bacterial viruses). Benzer had been pursuing a line of research aimed at determining the structure of genes. He was thus approaching the same problem that Crick and Watson had been pursuing, but Benzer worked from the angle of genetic analysis in the tradition of Morgan's fly lab, albeit a finer-grained form applied to individual nucleotides, whereas Crick and Watson sought a structural chemistry explanation. Benzer had happened upon an experimental technique that allowed him to isolate the precise location of genetic mutations on a section of a bacteriophage chromosome, the rII gene. His technique could identify locations with resolving power sufficient to locate individual nucleotide pairs.[36] He had been working on mapping the rII genes in detail. His "Fine Structure of a Genetic Region in Bacteriophage,"[37] published in 1955, followed Crick and Watson's paper on DNA by two years. Benzer's study showed that the rII gene mutations occur at sequential locations consistent with the linear arrangement of bases hypothesized by Crick and Watson. His work thus provided timely experimental support for Crick and Watson's theory, [38] as did similar work by Milislav Demerec.[39]

Benzer referred to his map of the location of mutations in the rII region as a topology, a map showing the spatial relationships between genetic material. In the case of DNA sites, the map is linear. Benzer followed his topology with an analysis of mutation rates -- that is, a study of the rates of occurrence of the mutations he had isolated in his topology. He referred to this latter analysis as a topography. The topographical map invoked the visual image of a histogram plot to provide the elevations of genetic change to go along with the locations of genetic data on the topological map.

Benzer's topographical map was an echo of Morgan's analogy of travel time between the waypoints on a train route where elevated rates of mutation at some sites represented the variations in mutation rates among DNA locations like the variations in travel time between equidistant waypoints. For a given DNA site, the topography showed the rate of change for the particular bit of DNA data located at that site. Whereas the topology showed the waypoints on the DNA map, representing individual DNA bases, the topography showed their pattern of change in a given unit of time, or more specifically for a given number of replications. This latter study, "On the Topography of the Genetic Fine Structure,"[40] published in 1961, sought to answer the question "are all the subelements equally mutable? If so, mutations should occur at random throughout the structure and the topography would be trivial."[41]

The anomalous result was a topography that was far from trivial. The rates of spontaneous mutation are highly non-random, contrary to the expectation from the DNA structure. Some sites were dubbed "hot spots" for their exceptionally high rates of mutation. Moreover, when compared to mutations induced by human intervention through chemical mutagenic agents, the pattern of spontaneous mutation was significantly different. Whereas the topography should have been characteristic of a simple Poisson process where each site was

equally likely to mutate, it was in fact an intricate landscape with irregular peaks and valleys representing unexpected persistent hot spots of varying magnitudes as well as cold spots.

Benzer did fit his data to a Poisson distribution as a way of estimating the number of sites with no mutations, which would not otherwise be apparent from his technique. Although he did not perform a formal goodness of fit test, the fact that he fit his data to the Poisson reflects an expectation that theoretically mutation should be equally likely at each site. When the possibility is considered that Benzer was observing raw divine action, unmediated by any natural cause, his explanation of the anomaly is earth shattering: "...the distribution of repeats is far from random. The topography for spontaneous mutation is evidently quite complex, the structure consisting of elements with widely different mutation rates."[42] This is an apt description for the methodical action of a divine craftsman fashioning new living beings; not so for the blind action of random chance.

Benzer, who expressed a complete lack of interest in religion from childhood,[43] was unlikely to see miraculous causes behind his results. He attempted a naturalistic explanation. Benzer speculated that genetic sequences that were higher in AT content would be more mutable due a weaker pair bond between the bases as compare to GC.[44] Could this simple bimodal effect explain the high level of variability in mutation rates among DNA sites in his reported data, particularly in light of the factors isolating individual base pairs from each other mentioned previously? In a letter to Sydney Brenner he emphasized the perplexity of the nonrandom pattern in apparent contradiction to his simple pair-bonding explanation: "But the mutation rates still make no sense in terms of simple-minded ideas (i.e. without recourse to very long range 'paragenetic resonance' or 'benzerine')."[45] Benzer's work predated the discovery of the genetic code as well as later gene sequencing technology. He did not know the DNA sequences behind his mutation rates. Francis Crick acknowledged the anomalous mutation pattern in a letter of his own to Sydney Brenner after a 1956 conference he attended where Benzer presented his findings up to that point: "However, the mutation behaviour does *not* fit the [Watson-Crick] simple mechanisms. In general the back rates [that is, the frequency of reversions from a mutant to the wild form] appear too fast, and the observed rates are all over the place."[46] Horace Judson further comments, "Crick tried out a couple explanations -- unconvincing ones that signified only that he had begun to give thought once more to the mechanisms of mutation."[47]

Benzer's work with the humble bacteriophage was published over fifty years ago. In the intervening period, mutation rates have been studied for all sorts of life. The DNA code is universal to all higher plants and animals.[48] James Watson, in his book *DNA: The Secret of Life* captured this observation succinctly: "a piece of DNA after all is finally still DNA, its chemical properties the same irrespective of its source."[49] In spite of the universality of DNA to all life, a modern genetics textbook notes, "the [spontaneous mutation] rate varies considerably among different organisms", and "even within the same species, the spontaneous mutation rate varies from gene to gene."[50] Neither effect is expected given the omnipresent characteristic DNA structure among different organisms and among individual genes within a particular species. Moreover, the same nonrandom pattern first seen in bacteriophage has also been observed in mammals.[51] The nonrandom pattern of spontaneous mutation by DNA site is as universal as the DNA code itself. What's more, nonrandomness by DNA site has been observed for chromosome crossover recombination events, just as it has for mutation, as noted earlier.

James Watson remarks on the phenomenon of site-specific spontaneous mutation bias in his classic text on the gene: "The overall rate at which new mutations arise spontaneously at any given site on the chromosome ranges from about 10^{-6} to 10^{-11} per round of DNA replication, with

some sites on the chromosome being 'hotspots' where mutations arise at high frequency and other sites undergoing alterations at a comparatively low frequency."[52] There is nothing in the structure of the DNA to account for this variability. Yet the magnitude of the bias is large, as Watson's text acknowledged: "Thus, an average nucleotide is likely to be changed by mistake only about once every 10^9 times it is replicated, although error rates for individual bases can vary over a 10,000 fold range."[53] Watson's characterization of these mutations as mistakes and errors reflects his commitment to the prevailing naturalistic paradigm of science.

Despite the ubiquity of site-specific mutation bias throughout DNA, science has only offered partial explanations for such phenomena. Watson's text cites the example of the DNA sequence CA repeats: "The replication machinery has difficulty copying such repeats accurately, frequently undergoing 'slippage'."[54] This is a possible partial account for one type of insertion or deletion mutation, but does not amount to an explanation for pervasive mutation bias. Absent a description of the "slippage" mechanism, it amounts to empty hand-waving. Explanations like this one and Benzer's invocation of a relatively weaker AT pair bond propose to explain some cases, but there are numerous anomalies and unexplained observables that remain unaccounted for by any comprehensive theory. These are the kind of "partial fit" explanations that Cornelius Hunter was talking about in the context of applying naturalism to a phenomenon that defies explanation in naturalistic terms.

Watson, an atheist,[55] would presumably be no more open to a miraculous cause than was Benzer or Morgan. Nor would Francis Crick, who, like Morgan cited his atheism as a motivation for his work in biology, in a conversation reported by Horace Judson: "An important reason Crick changed to biology, he said to me, was that he is an atheist, and was impatient to throw light into the remaining shadowy sanctuaries of vitalistic illusions."[56] The biologists closest to the discovery of the processes which originate genetic novelty were firmly wedded to the naturalistic scientific paradigm.

Only if the requirement that all phenomena be explainable in naturalistic terms is relaxed, is it possible to see that the observational data favor a miraculous cause. Absent the ideological commitment to philosophical naturalism, the empirical data favor an external nonrandom cause over an intrinsically random one. The data favor Asa Gray's theistic evolution hypothesis with a purposeful, nonrandom source of variation over the naturalistic evolution hypothesis of the neo-Darwinian synthesis with its random variation. The empirical evidence from the creative engines of evolution, namely, genetic mutation and recombination, support, rather than refute the existence of God.

An alternative picture to the Darwinian one emerges where God is intimately involved in evolution. Our parents are only conduits for the traits we acquire through inheritance. Ultimately, according to evolutionary theory, all traits initially arise spontaneously. In this alternative picture, God is operative in all aspects of evolution. God is the primary cause of all mutations. These are in turn inherited from our parents as the secondary cause. Some few of our traits are initially created in us directly as new or recurrent spontaneous mutations. Most traits are created in an ancestor in a prior generation and then passed on to us through our parents. Even for those traits inherited from our parents, therefore, God was the primary cause in a prior generation. Further, genetic recombination, like mutation, is a nonrandom process. Among our parents' traits, God chose the particular combination we would inherit, a personal example of unmediated divine action in everyone's conception. Ultimately, therefore, in this view, all of our traits come to us from God. That makes us all children of God.

[1] Edward J. Larson, *Evolution* (New York: Modern Library, 2004), 85.
[2] Charles Darwin, *On the Origin of Species* (New York: Penguin, 2009).
[3] Ernst Mayr, *What Evolution Is* (New York: Basic Books, 2001), 96-98.
[4] Douglas J. Futuyma, *Evolutionary Biology, Third Edition* (Sunderland: Sinaur Associates, 1998), 283.
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