Why Evolution Has Failed the Sciences

The Molecular Machinery of Repair and the Case that Life Was Designed Not to Evolve

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Abstract

The Neo-Darwinian synthesis asks us to accept that the biological distance separating humanity from an apelike common ancestor was crossed by nothing more than the accumulation and fixation of random genetic mutations, sifted across deep time by natural selection. This paper contends that the molecular evidence points decisively in the opposite direction. Rather than exhibiting the mutational openness that Darwinian change demands, the living cell is equipped with an elaborate, multi-layered apparatus whose measurable and undisputed function is to detect, excise, and reverse the very genetic alterations that evolution requires as its raw material. Over a dozen distinct DNA repair pathways operate continuously in somatic and germ line cells alike, correcting the overwhelming majority of the tens of thousands of lesions each cell sustains daily. The double helical architecture of DNA is itself a repair-ready design, storing two complementary copies of every base so that a damaged strand can be restored from its intact partner. Where a chromosome lacks a homologous partner, as with the male-specific Y chromosome, the genome supplies an ingenious alternative in the form of internal palindromic self-repair. I argue that a system engineered so comprehensively to prevent heritable change cannot plausibly be the engine of unlimited heritable change. The number of coordinated beneficial mutations required to convert an ape-like creature into a human being is so vast, and the waiting times so prohibitive, that molecules-to-man evolution is not merely improbable but effectively impossible. Life, in short, was designed not to evolve.

Keywords: *DNA repair, mutation, genetic entropy, common ancestry, intelligent design, Y chromosome palindromes, waiting time problem*

1. Introduction: The Claim on the Table

Evolutionary biology teaches, in its most candid form, that monkeys became men. The mechanism proposed is the preservation, by natural selection, of rare beneficial genetic mutations across an immense span of time. Recognizing that the blunt phrasing “monkeys became men” strikes most people as implausible on its face, its defenders have refined the presentation. We did not evolve from a monkey, they now insist, but rather humans and modern apes descended from a shared common ancestor. This is offered as though it dissolves the difficulty. It does not. The common ancestor in question is not some neutral, unspecified organism. On the standard reconstruction, it was an apelike hominid, and the anatomical and behavioral reconstructions that accompany it describe a creature far closer to a chimpanzee than to a human being. Relabeling the ancestor does not shrink the gap that must be crossed; it merely relocates the starting line.

The relevant question, then, is entirely quantitative and mechanistic. If an ape-like, chimp- like ancestor is to be transformed into a modern human, how much heritable change is required, by what molecular means is that change supposed to be introduced and preserved, and does the cell, as we actually observe it operating, cooperate with that program or resist it? This paper takes up each of those questions in turn. My central thesis is straightforward and, I will argue, decisive: the cell does not cooperate with the evolutionary program. On the contrary, the best documented feature of the genome relevant to this debate is a comprehensive, redundant, and demonstrably purposeful machinery whose function is to prevent heritable genetic change. Life was designed not to evolve.

2. The Size of the Genetic Gap

The popular figure, endlessly repeated, holds that humans and chimpanzees are roughly 98.8 percent identical, differing by only about 1.2 percent of their DNA. This statistic is quoted so often that it has acquired the status of settled fact, yet two considerations must be kept in view. First, even accepting the conventional 1.2 percent, the arithmetic is sobering rather than reassuring. The human genome contains approximately 3.2 billion base pairs. A 1.2 percent single-nucleotide difference, therefore, represents on the order of thirty-five million individual base pair differences, and once small insertions and deletions are counted, the number of nucleotide positions that differ climbs well past ninety million. The gap is not measured in a handful of tweaks; it is measured in tens of millions of specific, coordinated genetic changes, every one of which must arise, must not destroy function, and must be preserved.

A clarification of terminology is warranted here because the difference is sometimes described loosely as involving millions of genes. That phrasing should be avoided, since the human genome carries only about twenty thousand protein-coding genes in total. The correct and far more damaging way to state the point is that the divergence spans tens of millions of nucleotide positions distributed across those genes and the vast regulatory landscape between them. The precision matters because the evolutionary claim is not that a few genes were edited but that an ocean of individual base-level changes was navigated, blindly, without foresight, and without ever passing through a non-viable intermediate.

In addition, the 1.2 percent figure itself rests on methodological choices that inflate the appearance of similarity. When Tomkins (2013) re-analyzed the full chimpanzee genome against the human genome using more complete alignment methods, rather than the selective pre-filtered comparisons that generate the familiar figure, the average genome-wide similarity fell dramatically, to roughly eighty percent by his accounting. Whether one accepts the higher or the lower estimate, the direction of the correction is unfavorable to the evolutionary narrative: the true distance between the two genomes is larger, not smaller, than the marketing figure suggests. And the larger that distance, the more improbable its unguided traversal becomes.

3. The Nature of Mutations: The Engine that Runs Backward

Mutations come in three broad classes with respect to fitness: the beneficial, the neutral, and the deleterious. The distribution among these classes is radically lopsided. The great majority of mutations that have any measurable effect on fitness are harmful, and beneficial mutations, mutations that add new, functional, integrated genetic information, are vanishingly rare (Eyre, Walker and Keightley, 2007). Kimura's own neutral theory, developed to rescue molecular evolution, did so precisely by proposing that most substitutions are selectively invisible, which concedes that they contribute nothing to the construction of novel adaptive complexity (Kimura, 1968).

The evolutionist, therefore, faces a cruel accounting problem. To build a human from an ape-like ancestor, natural selection must locate and fix an enormous number of the rarest category of mutation, the beneficial and coordinately functional, while the far more numerous deleterious mutations accumulate faster than selection can purge them. This is the phenomenon that Sanford (2008) has termed genetic entropy: because slightly deleterious mutations are effectively invisible to selection, they drift into the population and accumulate generation upon generation, degrading the genome rather than improving it. The observed direction of genomic change over time is decay, not construction. An engine that runs backward cannot be the engine that built us.

The magnitude of the required construction is staggering. By Bergman's estimate, converting an ape-like creature into a human being demands on the order of 450 million favorable mutations, each arising, each avoiding lethality, each conferring, or at least not destroying, advantage, and each becoming fixed in the population. Set against a measured de novo mutation rate of roughly seventy new mutations per individual per generation (Kong et al., 2012), the overwhelming majority of which are neutral or harmful, the numbers do not merely strain credulity; they break it. There is not enough time in the assumed evolutionary history, indeed not enough time in a billion years for the required favorable changes to be found and fixed in sequence.

4. The Waiting Time Problem and the Cost of Selection

The impossibility can be made rigorous. Haldane (1957) showed, in what became known as Haldane dilemma, that there is a hard limit on how rapidly a population can substitute beneficial mutations, because each substitution imposes a reproductive cost that the population must pay. Given realistic population sizes and generation times for the supposed human lineage, the number of beneficial substitutions that could have been fixed in the available time is small, on the order of a few thousand, nowhere near the millions upon millions the transformation requires. ReMine (1993) developed this constraint at length and demonstrated its lethal implications for human origins.

More recently, Sanford and colleagues modeled the waiting time directly. Sanford, Brewer, Smith, and Baumgardner (2015) simulated a model hominin population and asked how long it would take for even a single modest string of coordinated nucleotide changes, the kind required to establish a new functional element, to arise and spread. The answer, even under assumptions generous to evolution, ran to tens or hundreds of millions of years for a single such feature. Since human-specific biology requires not one but an immense catalogue of such coordinated changes, the waiting time problem alone is fatal. The prehuman cannot survive, unchanged and waiting, across the geological ages required for its own genome to assemble the necessary edits. The intermediate is not viable, and the clock does not run long enough, regardless.

5. The Decisive Evidence: A Genome Engineered Not to Change

The foregoing establishes that evolution is improbable to the point of impossibility. But the positive case is stronger still, and it is this positive case that constitutes the heart of the argument. If we ask if the cell is actually built to utilize mutations, the answer is unambiguous: it is built to eliminate them. Each human cell sustains an astonishing burden of DNA damage, on the order of tens of thousands of lesions per cell per day, arising from spontaneous chemical decay, replication errors, and environmental insult (Lindahl, 1993). If this damage were allowed to stand, heritable change would indeed be rampant. It is not allowed to stand. The cell deploys an armamentarium of repair pathways that together correct the overwhelming majority, upward of 99.9 percent, of all such lesions before they can be fixed as mutations.

These pathways are not a single crude mechanism but a coordinated suite of specialized systems, each tuned to a particular class of damage. Base excision repair removes small chemically altered bases. Nucleotide excision repair excises bulky, helix-distorting lesions. Mismatch repair proofreads newly replicated DNA and corrects mispaired bases, raising replication fidelity by orders of magnitude.

Homologous recombination and non-homologous, end-joining repair the most dangerous lesions of all: double-strand breaks. Photoreactivation, direct reversal, translation systems, and further specialized pathways round out a defense in depth (Friedberg et al., 2006). The existence of over a dozen such mechanisms, operating not only in body cells, but critically in the gametes, the sperm and egg that carry heredity forward, is precisely what one would not expect if the germ line were meant to be a fountain of evolutionary novelty. One does not install a dozen firewalls around a system whose purpose is to burn.

The very structure of DNA testifies to this repair-first design. The molecule is a double helix, and the genetic message is written twice, once on each of the two complementary strands. When one strand is damaged, the complementary strand retains an intact copy of the information, and the repair machinery uses that undamaged template to restore the correct nucleotide sequence to the injured strand. This is not an incidental byproduct of chemistry; it is the functional logic of a backup system. A molecule tasked with faithfully preserving information across generations was given a built-in second copy for exactly the contingency damage that would otherwise corrupt it. The double helix is, in engineering terms, a redundant, error-correcting, information store.

6. The Y Chromosome: Self-Repair Without a Partner

A particularly striking confirmation of the design interpretation comes from the male-specific Y chromosome. The elegance of complementary strand repair depends on having a partner chromosome to supply an intact template. Most chromosomes come in homologous pairs and enjoy this advantage. The Y is the conspicuous exception: across most of its length, it has no homologous partner with which to recombine, and it should therefore, on any naive account, be uniquely vulnerable to irreversible mutational decay. It is not. The genome solves the problem with remarkable ingenuity.

The Y chromosome is loaded with massive palindromic sequences, stretches of DNA that read the same forward and backward, so that the chromosome carries internal mirror image copies of its own critical genes.

When a gene in one arm of a palindrome is damaged, the intact mirror copy in the opposing arm serves as the template, and the chromosome recombines with itself to repair the lesion through gene conversion. Rozen and colleagues (2003) documented abundant such intrachromosomal gene conversion across the palindromes of human and ape Y chromosomes, This shows that the Y is not a decaying relic as some claim, but a self-correcting structure.

Confronted with the one chromosome that lacks the ordinary repair partner, we find not neglect but a custom made engineering solution, redundancy built directly into the chromosomes own sequence. This is the signature of foresight, not of accident.

7. Conclusion: Evolution as Delusion

The convergence of several lines of evidence against evolution is difficult to escape. The genetic distance between an apelike ancestor and a human being is enormous, spanning tens of millions of coordinated nucleotide changes and, on the fuller genomic comparisons, larger still than the popular figure admits. The only proposed mechanism for crossing that distance, mutation filtered by selection, runs overwhelmingly toward damage and decay rather than toward the construction of new, integrated information. The waiting times required to fix even modest coordinated changes exceed the ages available, and the cost of selection caps the achievable rate far below what the transformation demands. And, most tellingly of all, the cell is observably engineered not to permit the very changes evolution requires, guarding its information with a redundant double-stranded design, over a dozen repair pathways operating in the germ line itself, and, where an ordinary partner is lacking, an ingenious palindromic self, repair system on the Y chromosome.

The fact is that life was designed not to evolve. The dozen DNA repair mechanisms present in every cell, including the gametes, exist to counter DNA damage and prevent mutation, and consequently, to prevent evolution. This system functions by design to reduce the random genetic variety that is supposed to be the engine of Darwinian change. The design of such a complex, redundant, and purposeful system is obvious to any observer not committed in advance to denying it. Converting a monkey into a man requires an enormous number of favorable mutations that will never occur in a billion years, and the machinery of the cell is arrayed against even the first of these mutations. Molecules-to-man evolution is not science but an illusion, and, pressed further, a delusion, one that a great many churches have unfortunately embraced. It is time for the sciences and for the church to recognize the evidence for what it plainly shows.

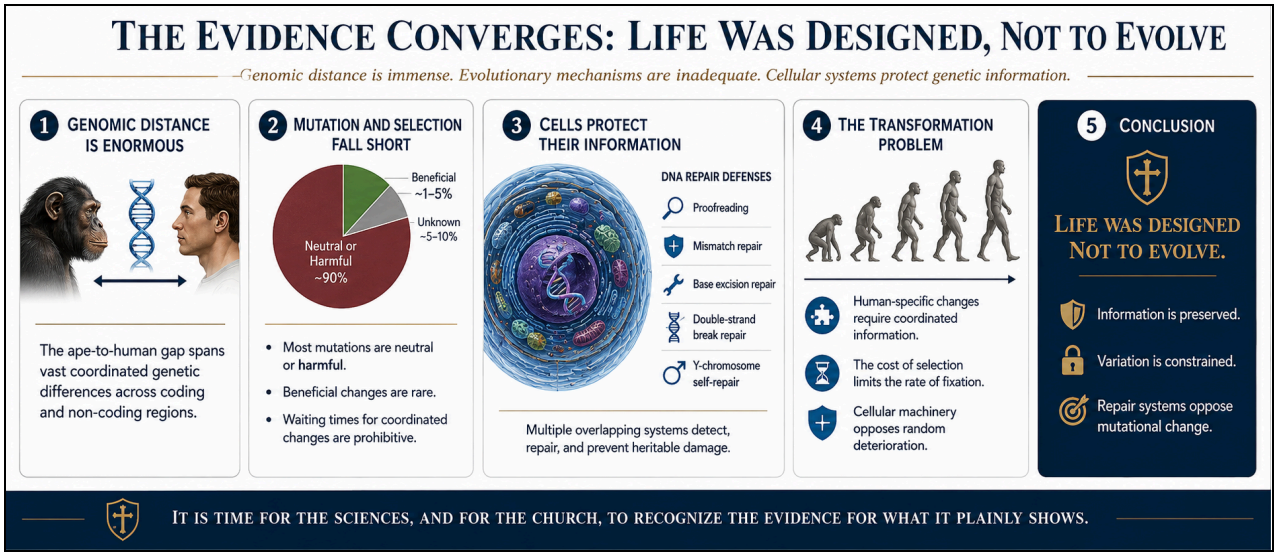

Figure 1. *Summary of the paper's argument that life was designed rather than produced by evolution. The diagram argues that the genetic differences between apes and humans are very large, helpful mutations are uncommon, cells contain systems that protect and repair DNA, and many major biological changes would need to happen together. Based on these points, the authors conclude that living things were designed to preserve genetic information rather than gradually transform into completely different forms.*

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