Healthy genomes require recent creation

Alex Williams

Every organism alive today has had many generations of ancestors, and natural selection has acted upon ancestral variation and *de novo* mutations to adapt them to changing environments. As a result, fine examples of intelligent design like the bacterial flagellum and the peacock's tail can appear diminished by an apparent 'evolutionary continuum' when examined in the context of their many relatives and likely ancestral species. Darwinists naively claim these outcomes as random products of nature. To expose this groundless assumption I here take a different approach by looking at the decay of design. When viewed against the black backdrop of genome decay and species extinction, the designs of life emerge triumphant in what Darwinists have universally taken for granted—healthy survival. Healthy genomes, a concept underlying both genetics theory and medical practice, provide an irrefutable 'engineering timescale' argument for both intelligent design and recent origin. Genome decay projected forwards points to extinction in just thousands of years, and projected backwards it produces perfect copy fidelity in the very recent past. Darwinism is emphatically denied. Only Genesis-style fiat creation can explain it.

Many arguments and evidences for the intelligent design of life have been put forward, but they have gained little ground in the public consciousness. Counter-arguments from Darwinists continue to blind the eyes of the majority and the challenge remains for creationists to do better. To this end we need to look at the decay of design—and in particular at genome decay. This phenomenon is not only acknowledged by Darwinists (albeit sometimes unwittingly), it is indelibly written into genetics theory, and it forms the essential backdrop to understanding medical genetics. The consequences profoundly refute Darwinism, and they make Genesis-style recent creation undeniable.

The neo-Darwinian genome

Darwinists believe that evolution on the grand scale is a fact:

> "As *a well-established scientific fact*, biological evolution still provokes heated debates all over the world about its compatibility with religious beliefs. … both *the scientific fact of evolution* and the Darwinian theory are concerns of philosophy and theology … [emphases added]."[1]

Such unquestioned allegiance is almost universal today.

Darwin himself imagined that every slightly beneficial variation would be naturally selected, leading to improvement in the owner and its offspring, and that all such changes would work together in a continuously onward and upward direction:

> "As all the living forms of life are the lineal descendants of those which lived long before the Cambrian epoch, we may feel certain that the ordinary succession by generation has never once been broken, and that no cataclysm has desolated the whole world. Hence we may look with some confidence to a secure future of great length. And as natural selection works solely by and for the good of each being, all corporeal and mental endowments will tend to progress towards perfection."[2]

With the discovery of mutations and the new science of genetics in the early 20th century, Darwinism needed a new formulation. English statistician R.A. Fisher provided it in his 1930 book *The Genetical Theory of Natural Selection* in which he outlined what became known as the Modern Synthesis (what we usually call neo-Darwinian theory). Although his only data consisted of 500 deleterious mutations documented in the fruit fly *Drosophila*, he proposed that mutations of any kind had some chance of becoming beneficial to their hosts if they only produced small changes. This foundational assumption resulted in an exponential distribution of fitness effects (figure 1), which became the standard expectation in all subsequently genetics studies.[3,4]

When the structure of DNA and its implied information coding system was unveiled by Watson and Crick in 1953, Fisher's model was given a more concrete application. It now said, in effect, that any kind of mutation at any point on the DNA molecule had a finite chance of being beneficial if its magnitude of phenotypic change is small. Darwin's belief that every slightly beneficial variation would be naturally selected seemed to have been affirmed, and the neo-Darwinian genome became an indefinitely mutable entity. Fisher's theory now provided a clear foundation for the Darwinian belief that everything had evolved from something else.

If every form of life evolved from some other form of life, then every genome must, in principle at least, be

capable of being transformed into anything else. I shall here apply this principle to the human genome in relation to the great apes. If neo-Darwinian theory is correct, the human genome must be capable of having been transformed from a common ancestor with the chimpanzee and gorilla within the last 10 million years.[6]

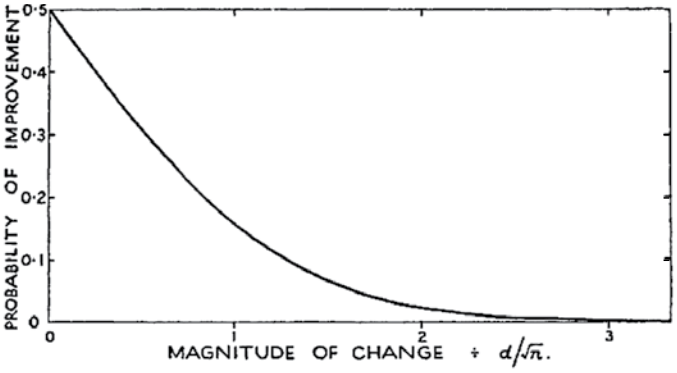

Fig. 3. The relation between the magnitude of an undirected change and the probability of improving adaptation, where the number of dimensions (n) is large

Figure 1. R.A. Fisher's 1930 graph and caption for the magnitude of change produced by a mutation of any kind and the probability that it will improve the species' chances of survival.[5]

Genome in crisis

Orthodox Darwinists can only appeal to random changes and natural selection. But mutations that occur in genomic regions which affect conserved core structure are highly likely to kill their hosts. The only theory that has been put forward as a comprehensive solution to this challenge of why mutations do not destroy life is what molecular systems biologists Marc Kirschner and John Gerhart[7, 8] called *facilitated variation theory*.[9] They noted that conserved core functions are modular, with 'weak linkage' between them. They likened the modules to Lego™ blocks that can be pulled apart and put together in different ways. The 'weak linkage' is provided by regulatory switching circuits that can be easily reorganized without damaging or changing the functional modules. As a result of this modular structure, natural variation would be built into the organism. Mutations and genetic rearrangements merely trigger into expression what is already present in potential. This creates a tremendous problem for evolution because it sets strict limits to natural variation and turns the 'tree of life' into a forest.[10] Thus, this theory hands creationists a ready-made explanation for within-kind variety.[11] It also provides built-in limits to natural variation, and allows very short timescales for diversification.[12]

The medical genome

Neo-Darwinian theorists have always relied on a computational approach to mutations, but medical doctors have to deal with mutations in real life. Down syndrome was one of the first genetic disorders to be described in detail.[13] The cause—an extra copy of all or part of chromosome 21—thus constituted a pioneering result in medical genetics: a healthy human genome should only contain 23 homologous pairs of chromosomes, one complete set from the mother and one complete set from the father.

With the advent of DNA sequencing technology we are today discovering thousands of new genetic disorders every year.[14] Table 1 lists the numbers of genetic diseases (in rank order) published at the time of writing, totalling 156,932. Compare this to the tally for single nucleotide variations (SNVs, the simplest kind of mutation): 88 million.[15] An internet search on the topic of beneficial mutations in humans produced barely enough to count on one hand, and most of them were side effects of deleterious changes. The most promising was the claim that Tibetans had evolved an increased ability to absorb oxygen in the thin air of the Tibetan plateau, compared to Han Chinese. This conclusion was short-lived, however, since a subsequent study showed that the genetic signature had been inherited from ancestral Denisovans.[16]

According to the *1000 Genomes Project* the average number of SNVs per person today is 3.6 million.[18] A subsample of 179 healthy individuals found them to be carrying about 400 'disease associated' mutations and 2 'disease

Table 1. List of disease-causing mutations from the Human Genome Mutation Database maintained by the Institute of Medical Genetics in Cardiff, Wales.[17] Every known kind of mutation appears in this list.

Type of Mutation	Number
Missense/nonsense SNVs	87,173
Small deletions	23,731
Splicing	14,302
Gross deletions	11,683
Small insertions	9,917
Regulatory	3,024
Gross insertions	2,797
Small indels	2,282
Complex rearrangements	1,567
Repeat variations	456
Total disease-causing mutations	**156,932**

causing' mutations.[19] Mutation databases are only recording 'deleterious' and 'functional' categories—there is no 'beneficial' category.[20–22]

Whole genome measurements of human mutation rates suggest a value in the region of 40 new SNVs per person per generation,[23,24] while a combination of methods suggests the rate is ~70.[25] Today we know that our genomes are 'full of functional elements'[26] and they are 'pervasively transcribed',[27] so it is likely that no mutation is truly neutral and all are likely to be at least slightly deleterious. A 'functional' mutation would thus be damaging, but not damaging enough to destroy function—although there is some controversy over the meaning of the word 'function'[28] in this context. A very telling argument on this point is that table 1 shows that *every known kind of mutation causes disease*. A recent review concluded:

> "Finally, we examine models involving slightly advantageous mutations. We show that the distribution of the absolute strength of selection is well estimated if *mutations are assumed to be unconditionally deleterious* [emphasis added]."[29]

Hereditary diseases

How do scientists decide when a genetic disorder is present? One indicator is heredity. Haemophilia—a defect in the blood-clotting mechanism that stops injuries from bleeding—figured prominently in the history of European royalty in the 19th and 20th centuries.[30] The mutation is recessive and occurs on the X chromosome. Females who carry one affected chromosome will not manifest the disease because the undamaged gene on the other X chromosome will produce the right protein for blood clotting. Males who inherit the damaged gene will normally manifest the disease because they have no undamaged gene to counteract it. However, there is also an anticoagulant system in the body that prevents clots from forming in inappropriate places, which would cause thrombosis. A male with a weakened anticoagulant mechanism will suffer less from inherited haemophilia than an otherwise normal male.[31] People with haemophilia B lack a protein called factor IX that is crucial for forming blood clots. Supplements of factor IX are currently used to treat it, but gene therapy is showing promise of a permanent cure.[32]

But more and more diseases today are turning out to have multiple genetic 'risk factors', sometimes hundreds of them. While many such diseases have a research history from study of families and twins, the latest methods included genome-wide association studies (GWAS). Disease complexes are identified by searching through the genomes of people with the disease compared with control subjects who do not have the disease symptoms. Examples of diseases with multiple genetic risk factors include diabetes, heart disease, schizophrenia, multiple sclerosis, Alzheimer's, Parkinson's, ADHD, gout, celiac disease, lumbar disc disease, bipolar disorder, asthma, allergic rhinitis, atopic dermatitis, autoimmune disorders, Crohn's disease, stroke, autism, lupus, Paget's disease, and more.[33]

So how do doctors know what a healthy genome looks like? One way is to look at the chromosomes—a healthy genome contains an even number of matching chromosomes;[34] one complete set from the father, and one complete set from the mother. Any other combination is likely to cause disease. And where did the gene therapy researchers get their DNA with a functional factor IX gene? From the appropriate section of an *undamaged* X chromosome belonging to a person who did not suffer from haemophilia. Doctors recognize that mutations damage genes and cause disease.

The natural genome

Darwinists pride themselves on appealing only to natural causes, but cells have so many mechanisms to detect and remove mutations (DNA damage) that they clearly see changes to these systems as *un*-natural and damaging. DNA repair is such a burgeoning field of research that it now has its own research journal.[35] Cells also recognize when mutation damage is beyond repair—they invoke a 'suicide' option called *apoptosis* to dismantle the cell and recycle its components. Cells thus exercise natural self-selection and eliminate damage caused by too much mutation. This is entirely consistent with what medical genetics tells us.

Natural variation cannot be equated with mutation. Natural variation is primarily caused by that which is built into sexually reproducing organisms—the homologous recombination that takes place during meiosis.[36] Mutations add to variation, and sometimes can create new traits that selection can then focus on (e.g. sickle cell anaemia, because it provides some protection against malaria). Yet, since mutations are highly correlated with disease, they cannot be the sole, or even a major, source of variation.

Genome decay

The fact that mutations lead to genome decay is now well established. Baer *et al.*[37] noted the widespread occurrence in multicellular eukaryotes of deleterious mutation rates greater than the threshold value of "one per generation" that would lead to "inexorable decay". Fisher should have realized it in 1930 when he gathered the available data on mutations and found that they were all deleterious. John Sanford's pioneering work,[38] which introduced the term 'genetic entropy', has now been supplemented with a number of other studies that confirm the devastating implications for

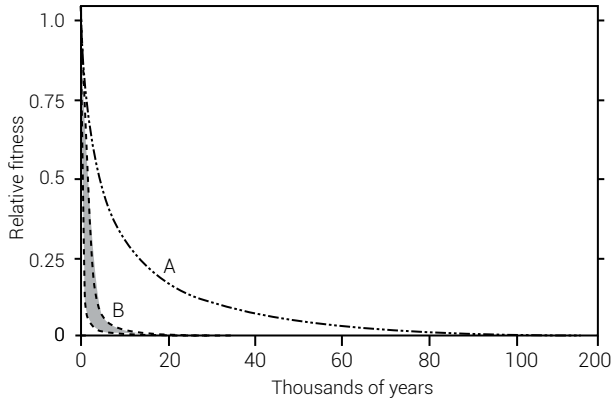

Figure 2. Human reproductive fitness decline due to genome decay. Curve A shows the rapid decline in fitness predicted by *Mendel's Accountant* using default human population parameters with today's mutation rate. The shaded region at B represents even more rapid fitness declines of 1% (upper curve) and 3% (lower curve) of today's values (per generation), as reluctantly admitted by leading genetics experts.

neo-Darwinian theory. The fundamental principle is easy to understand. Only lethal or strongly deleterious mutations can be removed from a population by natural selection. Slightly deleterious mutations are passed on. As they accumulate across generations, reproductive fitness declines and the species heads inevitably towards extinction. Numerous studies, using a variety of approaches, and all testifying to the same end, were published recently in *Biological Information: New perspectives*.[39] Most notably *Mendel's Accountant*, a comprehensive simulation of the fate of all new mutations in a population,[40] is proving to be an invaluable research tool. Even the digital evolution program *Avida* confirmed genome decay when biologically realistic data were used.[41]

The nearest that Darwinists have come to recognizing genetic entropy is in Muller's Ratchet—the inability of asexual species to remove deleterious mutations via meiotic recombination. But they believe the ratchet 'clicks' only when the least-mutant member of a population dies.[42] In reality the ratchet clicks multiple times every generation because a multitude of new mutations are added during each generation. Laurence Loewe's initiative was absurdly launched with the title "*Evolution@home*: Global computing quantifies evolution due to Muller's ratchet".[43] But, as is clear from the scientific record, the ratchet leads to extinction, not evolution. Loewe found that genomic decay in human mitochondria presented an evolutionary paradox, and he had to acknowledge that nuclear DNA was equally threatened with extinction, so he appealed to "unconventional explanations for long-term persistence".[44] He subsequently explored the problem in two asexual species,[45,46] then did a review of the field,[47] after which the subject disappeared from his CV.[48] His review included this observation: "one can argue that extinctions are always caused by a lack of mutations that enable adaptation".

To visualize the consequences of human genome decay, figure 2 shows the decline in reproductive fitness predicted by *Mendel's Accountant* using default human population parameters with the lower estimate of today's mutation rate (40 per person per generation). In addition there are two curves representing 1% and 3% fitness declines per generation which represent lower estimates of the impact of mutation load made (reluctantly) by leading geneticists.[49,50]

Genome copying fidelity and reproductive fitness

Figure 2 shows that humans cannot possibly have evolved from a common ancestor with the great apes over millions of years. If we had been around for that long we would have mutated to extinction many times over. But it also raises serious questions about the state of our genomes if our species has existed for even several thousand years. Curve A in figure 2 shows a fitness decline to almost 40% after 6,000 years—is this realistic? According to a recent global survey,[51] primary infertility (inability to have a first child) affects about 2% of the world population, and secondary infertility (inability to have a second or further child) affects about 10%. Higher rates are found among older couples. Clearly the models represented in figure 2 are overestimating the impact of mutations on reproductive fitness.

How might we arrive at more realistic estimates? One way is to consider the amount of variation built into the human genome in the beginning. Carter has argued, and I agree, that since apparently healthy people today are carrying millions of SNVs then we should expect that our (perfect) original ancestors also carried millions of healthy variations (polymorphisms), originally put in place to produce phenotypic novelty, partially for future adaptation to changing environments.[52] If that is the case, the number of deleterious mutations accumulated since creation can be drastically reduced. Is there a way to estimate the number? Yes, there is—we can project genome copying fidelity backwards to explore the past.

Copy fidelity measures the success rate of genome copying rather than the error (mutation) rate. It is most easily understood when expressed in fraction or percentage terms. Starting from a perfect state, and with a fixed copy fidelity of 90%, a genome would contain more nonsense than information after just 7 generations ($0.9^7 = 0.48$). It is obvious from these calculations that only very high initial copy fidelity could maintain a genome over many generations.

But does copy fidelity itself decay over time? Williams[53] recently explored the implications of a model with decaying copy fidelity for the origin of life. He demonstrated that life requires not only an extremely high standard of original

design and construction in its replication machinery, but also a high standard of ongoing maintenance and repair to sustain it over thousands of years. High genome copy fidelity requires the rapid and efficient detection and correction of copying errors and other DNA damage events. Many different systems are involved,[54] and new discoveries are continually being made.[55] Faithful replication of DNA support structures (e.g. histones) is also crucial to genome copy fidelity.[56] Here is a recent summary of the field of DNA repair:

> "DNA repair is responsible for preserving the genome of all cellular organisms. [It also] controls mutation rates that generate genetic variation in response to environmental changes. These conflicting tasks are finely tuned … [for] the difficult task of maintaining the proper balance of the entire repair system over a wide range of conditions … . Moreover, DNA repair needs to be coordinated with DNA replication, transcription, and chromosome organization processes, which can in turn be regulated by damage responses. Although the main repair pathways [are known] we are still far from understanding the overall organization of DNA repair … it is unclear how the different repair components cooperate to create functioning pathways, how the pathways coordinate and integrate with other cellular processes, and how environmental changes modulate the organization of the repair system."[57]

In short, we don't yet know whether copy fidelity is decaying along with the rest of the genome or not. Nor do we know what form it would follow even if it were decaying. For example, it might be collapsing exponentially, or it could be incrementally decreasing with each generation, or there may be a threshold (akin to the concept of mutational meltdown) where the fidelity slowly decays to a point beyond which it rapidly diminishes as the species approaches extinction.

But the following power function model allows us to explore copy fidelity in relation to the 'healthy genome' concept in a simplified manner. There are other possible scenarios, but they will all follow the same trajectory *in general*. Here is the basic equation:

$$Q_t = (Q_0)^n \quad (1)$$

where Q_t is the copy fidelity at time t (in generations), and Q_0 is the copy fidelity at some reference point n generations prior to time t. We don't know the exact contribution that copy errors make to our total mutation burden, but we can use the lower estimate from whole genome studies mentioned earlier (~40 per genome per generation) as a first approximation. We turn the mutation rate into a copy fidelity rate as follows: when projecting forwards, $Q_0 = 1 - (40 \div 3{,}000{,}000{,}000) = 0.999999987$ (or 99.9999987%). The same number can then be used as the value of Q_t when projecting backwards into the past (figure 3).

Equation (1) can be solved forwards by iterating numerically through generations and accumulating mutations until the current average mutation burden of 3.6 million SNVs per person is reached. But numerical iteration of equations containing numbers close to 1 or 0 can rapidly accumulate rounding errors, so the resulting value of n needs to be checked by substituting it into equation (1) and solving analytically for the number of mutations in the n^{th} generation. The result, using both methods, was n = 424 generations (~8,480 years) (figure 3). Calculations were carried out using *Mathematica* v.9.0.1 (64-bit version), and 50-decimal-place precision was required to eliminate rounding errors.

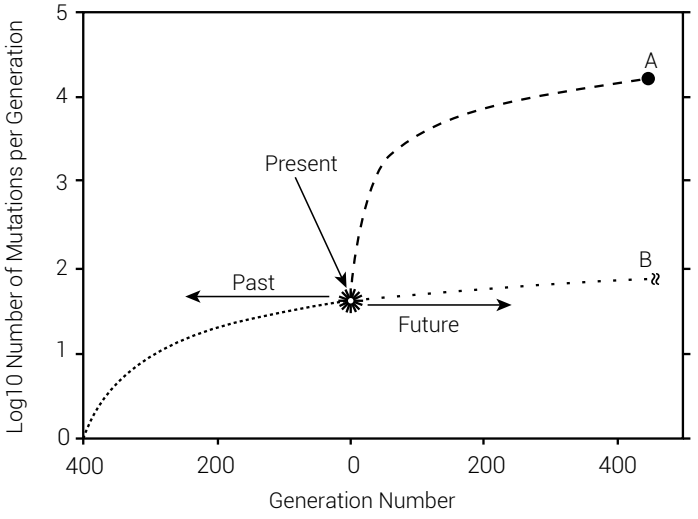

Figure 3. The behaviour of equation $Q_t = (Q_0)^n$ starting from today's mutation rate (the asterisk symbol, which represents 40 mutations per genome per generation). Forward projection (dashed curve A) accumulates the current mutation load of 3.6 million per genome after 424 generations (~8,480 years). The dotted curve is the trajectory followed if the 'zero error' primordial copy fidelity is projected from the past through the present and into the future (curve B).

Equation (1) can also be solved from some time in the past either iteratively by generation or by taking the n^{th} root of a value of Q_0 which reproduces today's mutation rate. At exactly 400 generations into the past a value of Q_0 equivalent to 1 error per 30 billion nucleotides per generation reproduced today's value of 40 copy errors per genome per generation. The error rate was so low at this earlier point that the genome would have been copied with 100% accuracy. Integrating the number of mutations produced in each generation over these 400 generations yielded a cumulative total of 8,020. This constitutes just 0.2% of today's mutation load of 3.6 million SNVs per person, so 'other causes'

must have contributed the remaining 99.8%. This means that the previous forward projection of equation (1) gives a very large overestimate of the actual time required because copy errors could not have contributed more than 0.2% of today's total mutation burden.

These considerations now give us a second way of projecting into the future. We can continue the primordial 'zero mutation rate' through today's mutation rate and then on into a future in which mutations accumulate at a much more slowly increasing rate. That is, copy fidelity remains much higher for much longer than in the earlier forward projection. In this case the model accumulated the present mutation burden in 8,485 generations (~170,000 years) from copy errors alone, which is again a very large overestimate because it ignores the major contribution from 'other sources' to today's SNV count.

If we follow Carter's argument we could say that the 99.8% of SNVs not attributable to copy error could be healthy polymorphisms that were present in our originating ancestors. Would this be more realistic? Perhaps, but we know that many kinds of events other than copy error can contribute damaging mutations to genomes, and we cannot know their precise history. Such events include ionising radiation from cosmic, solar, and local sources, toxins of many kinds in differing sources of food, air, and water, and reactive oxygen species that arise naturally in cellular metabolism and are not always promptly neutralized. Within the creationist model a hypothesized global period of accelerated nuclear decay is one potentially very large cause.[58]

Radiation therapists have long searched for ways to minimize radiation damage to healthy tissue. One enduring mystery has been that patients vary enormously in their tolerance of radiation, with 80–90% of the variation being unexplained by standardized tests on tissue damage.[59] The recent availability of whole genome sequencing has shed some light. A study of reactions (in this case, erectile dysfunction) to radiation treatment for prostate cancer identified twelve SNVs that lie in or near genes involved in normal erectile function or other normal cellular functions, rather than [as expected] in mechanisms associated with DNA damage repair.[60] They showed that the risk of developing erectile dysfunction increased by 2.2 times for each extra SNV in these already mutation-damaged regions of their genomes.

Geneticists make an important distinction between SNVs shared widely in the population and those that are rare, usually only occurring in one or a few people or in localized populations. It is the latter group that contributes most to the wide variation in individual response to radiation therapy.[60] We can quantify this difference by referring to the dbSNP database.[61] Figure 4 gives the frequency distribution (dark grey bars) of all named and numbered SNVs identified in the '1000 Genomes Project' (81 million).[62] The vast majority of these, 68 million, are rare, with frequencies in the range 0.0001 to 0.01, and half of these, 34 million, are in the rarest category of ≤ 0.0002. Among the common variants, 5.4 million fall in the range 0.11 to 0.5, and 13 million fall in the range 0.01 to 0.5. We can therefore reasonably accept Carter's conclusion: "I expect Adam had about 10 million or more heterozygous loci." Furthermore, only a total of 1,733 of these were identified as 'pathogenic' or 'likely pathogenic' (figure 4, light grey bars with numbers), which supports the idea that only a few thousand mutations have accumulated since creation.

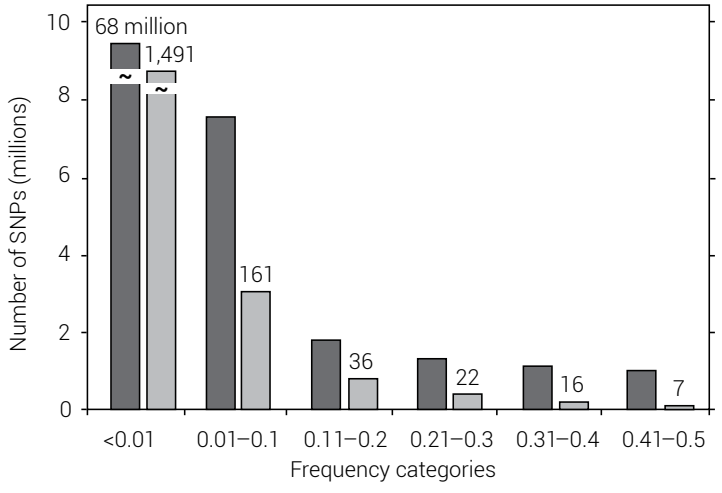

Figure 4. Frequency distribution of total SNPs in the '1000 Genomes Project' data (dark bars, left hand scale in millions) and the few identified as 'pathogenic' or 'likely pathogenic' (light bars with numbers). In both cases the most frequent are the rare ones (far left columns).

We can now use the infertility data to shed light on the rate of genome decay. Secondary infertility is the value relevant to species survival—mothers must, on average, have more than two reproductively viable offspring to ward off species extinction. So we can use the current secondary infertility rate (10%) to calculate our species' reproductive fitness after 6,000 years at 90%. Assuming fertility factors are heritable in the same way that copy fidelity factors are heritable, and assuming a power function like equation (1), we can calculate reproductive fitness decline over time. In this case Q_t = fitness at time t (in generations), and Q_0 = fitness in the first generation after the Fall.

The result of this calculation is $Q_0 = 99.966\%$. When converted to an infertility rate (for comparison with figure 3) it starts at zero and traces out a history that follows the shape of curve B, but with its origin at 300 generations

(6,000 years, assuming 20 years per generation). When projected into the future, infertility reaches 50% after 2,000 generations (40,000 years); beyond this point the population would rapidly decline to extinction.

We can also use *Mendel's Accountant* in a similar way to obtain a population-based estimate of the same history. A starting value of 5 new mutations per individual per generation, with the default human population values, is enough to reproduce today's infertility rate of 10% after 300 generations. During this period only 1,500 new mutations are accumulated. When projected into the future, infertility reaches 50% after about 12,000 generations (240,000 years), during which time about 60,000 mutations accumulate.

Discussion and conclusions

These modelling exercises suggest that among the millions of SNVs in human genomes, only a few thousand are needed to explain the current decline in human fertility. When projected into the future it seems that only a few tens of thousands of similar mutations will be enough to drive us to extinction. Such conclusions are devastating for Darwinism.

Geneticists have long known that they face severe problems. James Crow addressed the National Academy of Sciences on fitness decline through mutation accumulation and concluded:

> "I do regard mutation accumulation as a problem. It is something like the population bomb, but it has a much longer fuse. … the characteristic time is some 50–100 generations, which cautions us against advocating any precipitate action. We can take time to learn more."[63]

But what is there to learn? Crow's timescale (and those in figures 2 and 3) is disastrous for Darwinism. Gene therapy is making headway[64] but at present is too risky to use on germ-line cells (eggs and sperm). And because we all carry a multitude of mutations and have identified hundreds of thousands of genetic diseases it is hard to know where to start in repairing a mutation-damaged genome.

The negative impact of mutations is built into genetics theory. The 'selection coefficient'—the central parameter that implements Darwin's theory—is defined as the fraction by which a mutant is *less fit* than the wild type. Nobel Prize-winning geneticist H.J. Muller defined 'mutation load' as "the overall *reduction* in mean fitness relative to *the mutation-free genotype* brought about by *recurrent deleterious mutation* [emphases added]".[65] Darwinists should stop being double-minded about their own subject matter and listen to what the medical profession is saying—mutations cause disease! Genomes are healthy only in so far as they are mutation-free.

How healthy are our genomes today? Adam could have carried millions of healthy variations in his genome, and most of the millions we carry today could have been passed down to us unchanged. Deleterious mutations occurring after the Fall would seem to number only in the thousands. This makes the new mutation problem much more severe than previously expected. We are each carrying hundreds of mutations that have already degraded some of our organ systems to some degree, and the radiation therapy experiments show that every single new mutation doubles the risk of dysfunction to organ systems that are already damaged by mutations.

The models considered in this article all point to a primordial error-free 'healthy genome' just thousands of years into our past. There is no room anywhere—either in the experimental or theoretical data—for the Darwinian view of the human genome evolving 'upwards' over millions of years via mutation and natural selection. It simply does not exist. The inescapable conclusion is that humans must have been created with mutation-free 'healthy genomes' just a few thousand years ago, and their future is likewise limited to thousands, not millions, of years.

Acknowledgments

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Alex Williams *B.Sc., M.Sc.(Hons), M.AI.Biol., Dip.C.S., Th.L. has been a professional botanist, analytical chemist, environmental consultant, statistician, missionary, science writer, and illustrator, and has published numerous peer-reviewed articles on a wide range of subjects. He was an Australian representative and then consultant to the International Atomic Energy Agency, chairman of an international group of scientists, and delivered the invited review in his field at an international symposium. He is currently research associate at the Western Australian Herbarium in grass taxonomy, and has contributed many items to Creation and* Journal of Creation *and co-authored* Dismantling the Big Bang.