A successful decade for 'Mendel's Accountant'

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A powerful computer program with far-reaching consequences has been developed by a group of biologists and computer scientists. Striking at the heart of neo-Darwinian theory, it tackles the subject of mutation/selection using a straightforward method called genetic accounting. Named 'Mendel's Accountant', this software platform provides a comprehensive refutation of multiple aspects of evolutionary theory using nothing but standard evolutionary population genetics. The developers have used it to quantify the actual selection threshold for new mutations, to test alternate evolutionary ideas (e.g. unusual selection models, such as 'synergistic epistasis'), to quantify the long-standing 'waiting time problem' for new beneficial mutations, to make predictions about the long-term effects of mutation accumulation in viruses (which were later confirmed), and to compare different historical population models to the modern human mutation frequency spectrum seen in the 1000 Genomes Project data. Their results represent a complete refutation of the 'primary axiom' of neo-Darwinian theory. Computationally, the mutation/selection model fails in multiple ways.

After the success of Radioisotopes and the Age of the Earth (RATE),[1] the Institute for Creation Research began another major endeavour they called the GENE project. Even though this second project received much less fanfare, it achieved significant results and documented these in multiple publications. Essentially, every major goal they began with has been achieved. My involvement in the GENE project led to the publication of the 'mitochondrial Eve' consensus sequence in *Nucleic Acids Research* and a follow-up paper at the 2008 International Conference on Creationism.[2,3] But one aspect of the GENE project deserves special attention: the population-modelling program 'Mendel's Accountant'. This is a computer platform that enables users to test fundamental aspects of neo-Darwinian theory, using nothing but the tools of standard evolutionary population genetics. Major discoveries and advances were made possible thanks to the development of this powerful research tool.

Setting up the problem

For many years, neo-Darwinian population geneticists have been boasting about how successful their ideas were in making predictions, but they were obscuring significant problems. Specifically, they had grave mathematical difficulties when trying to explain how mutation and natural selection could produce the observed complexity of life. John Sanford calls mutation/selection the 'primary axiom' of neo-Darwinism in his groundbreaking book *Genetic Entropy and the Mystery of the Genome*.[4] In it, he says that population geneticists know that most mutations are deleterious. Also, it is widely known that natural selection and the occasional beneficial mutation cannot stop the gradual decline caused

by the accumulation of these slightly deleterious mutations. Individually, most mutations do no harm to the organism. But collectively, they can be catastrophic. They relentlessly accumulate in the genome over time because they are too weak to affect reproductive success. Since these deleterious mutations accumulate faster than they can be eliminated, the net result is genetic deterioration. When too many errors occur in a population, it enters a phase called 'mutational meltdown', which rapidly leads to extinction.

That is not merely a theoretical possibility. Given the measured rates of mutation and reproduction, Mendel's Accountant (the focus of this paper) shows that current evolutionary models result in genetic deterioration, suggesting that all complex life[5] is degenerating. This fundamental problem exposes the unreality of Richard Dawkins' popular (but misleading) computer program that generated the phrase 'Methinks it is like a weasel'.[6] Dawkins' simulation used an unrealistically low rate of mutation (merely one per progeny), plus an unrealistically high rate of reproduction (approximately 200 progeny per female), plus an unrealistically high rate of beneficial mutation (one in 27), and it locked any 'correct' mutation in a step-wise fashion, as if each step in the right direction was more grammatically correct than the nonsense phrase that preceded it. In these ways, and more, Dawkins' simulation was exceedingly unrealistic in favour of evolution.

The logic behind the genetic entropy problem was compelling, and was consistent with earlier mathematical analyses,[7] but it was largely theoretical in nature when Sanford first wrote *Genetic Entropy*. Empirical testing of genetic entropy theory only became possible when a team of computer scientists and geneticists developed a computer

simulation that could realistically and comprehensively model the mutation/selection process. The result was Mendel's Accountant. This program models the mutation/selection process based upon the conventional, textbook understanding of evolutionary genetics. Unlike other modelling programs of this type, the key parameters are adjustable—allowing the simulation and testing of nearly any scenario of interest.

The program has undergone multiple updates and improvements, and significant new features have been added over the years (such as dynamic population size modelling). Since population modelling uses tremendous amounts of computer memory, the latest version is designed to run on large servers.

How does Mendel's Accountant work?

Programs of this type need to be documented and validated in the professional literature. This happened in 2007 and 2008.[8–10] The program tracks all the mutations that occur among the individuals in a modelled population using a process the authors call 'genetic accounting'. When a simulated mutation occurs, it is assigned an identifier, a location on a chromosome, and a 'fitness' score using values that come from standard evolutionary genetics theory (i.e. although rare beneficial mutations do occur, almost all mutations are 'slightly deleterious'). By allowing for chromosomal recombination, the accumulating mutations get mixed into various combinations along each chromosome. Since children randomly inherit half of their father's and half of their mother's genome, some mutations are immediately lost. When it comes time to reproduce, some individuals fail to reproduce because they carry a greater load of harmful mutations. These are the individuals that are selectively eliminated, similar to the way Charles Darwin imagined natural selection to work. The program is extremely flexible, by design. Various models of selection, fitness, mutation, mating, and population structure can be applied to a given simulation.

What have we learned from Mendel's Accountant?

Early publications on Mendel's Accountant showed that, under normal conditions, mutations accumulate in a highly linear manner. This results in a continuous increase in the number of mutations per progeny over time. This increase is inexorable and irreversible, even when selection pressure is strong. As a result of the ever-increasing mutational burden, the Mendel simulations revealed that 'fitness' (a measure of total functionality) declines continuously over time, even with strong selection (figures 1 and 2).[11] This is a central problem for evolutionary theory, because if deleterious mutations cannot be removed, a species will deteriorate,
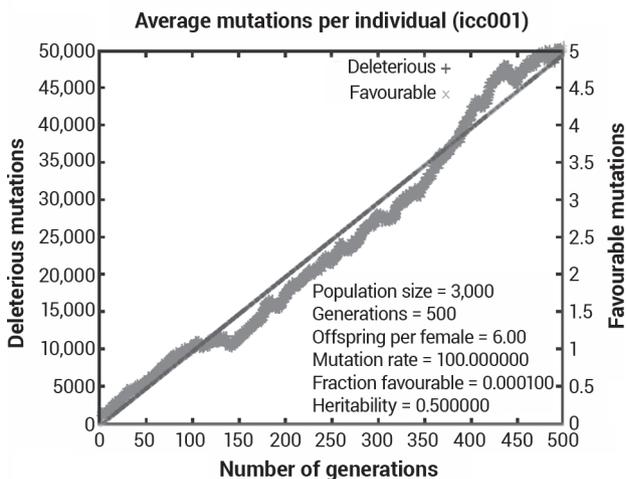

Figure 1. The number of deleterious and beneficial mutations accumulating in a model population over time, despite the winnowing effects of natural selection (after Baumgardner *et al.* 2008[10]). Straight line: deleterious mutations. Irregular line: favourable mutations. Since beneficial mutations are rare in standard mutation models (note the scale difference on the second y axis), random frequency fluctuations are more likely to affect the average. Thus, the favourable line has more 'wiggle'.
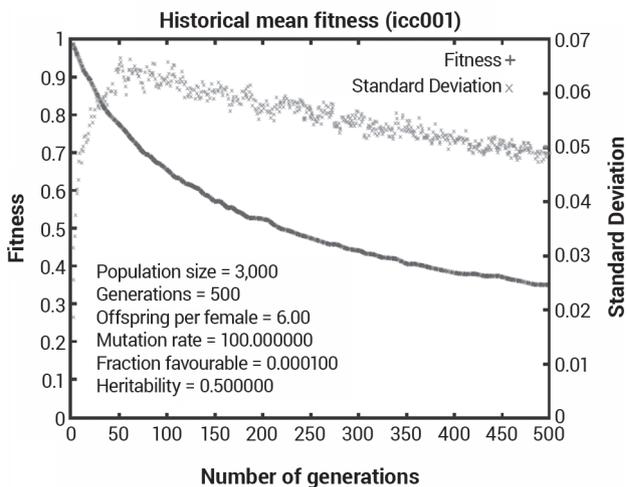

Figure 2. Inexorable decline in fitness (measured in terms of reproductive success) in the same model population as figure 1 (after Baumgardner *et al.* 2008[10]). Thick line: average fitness. Scatttered points: standard deviation of fitness at each time step.

all the way to the point of extinction. This is the essence of 'genetic entropy', and thus Mendel validates genetic entropy theory.

For the first time ever, we can now model how quickly mutations accumulate in large populations, the effects of natural selection, the effects of population bottlenecks, and what happens when rare 'beneficial' mutations appear, all thanks to this evolutionary modelling program.

A Mendel review paper appeared in 2012 as a chapter in an academic compilation of papers relating to population

genetics.[12] This chapter not only described how Mendel worked, but it also described how Mendel compared to other significant evolutionary modelling programs. As of the time of writing, it was the only biologically realistic program of its kind. This is still true today.

Mendel's Accountant is the most accurate software available for realistically simulating evolutionary genetic models. That has been true since its beginnings a decade ago. Why weren't evolutionists the ones to produce it, or improve on it? Evolutionists had the know-how, plus access to super computers, and public funding. So why did a group of what they consider outsiders produce such a program and publicly promote it?

The answer is illuminating. There existed primitive forerunners of the Mendel's Accountant simulation. These forerunners, created by evolutionists, were simple by comparison, yet when realistic values for human reproduction rate and mutation rate were used they were already demonstrating genetic deterioration. In other words, the problem of genetic deterioration was known long ago, both in theory and in computer simulations, but they failed to pursue it further, as least not publicly.

Even today, anyone might attempt to refute the results of Mendel's Accountant by producing a more realistic simulation. But they have not done so, not even in 10 years. This lack of serious response further suggests that Mendel's Accountant is demonstrating real problems for evolutionary theory.

Biological Information—New Perspectives

In 2011, a conference was held at Cornell University entitled *Biological Information—New Perspectives* (BINP). Multiple speakers from a wide range of backgrounds presented papers at this conference, and Mendel's Accountant was featured heavily. Indeed, one of the goals of the conference was to highlight its power, and six papers based on Mendel were delivered. Each paper is readily accessible online (see references).

A paper by Gibson *et al.* addressed the question of whether or not purifying natural selection could preserve biological information.[13] They concluded that selection was unable to preserve genetic information[14] because it systematically degenerates. Therefore, all genomes decay over time. Intense selection easily removed the worst mutations, but the mutations that were slightly deleterious accumulated relentlessly and without limit.

Sanford *et al.* empirically measured the threshold where selection breaks down (called the 'selection threshold') and determined what factors affect it the most.[15] In other words, they measured how impactful a mutation must be before natural selection can 'see' it. They found that almost all mutations (both beneficial and deleterious) sit below the selection threshold, and so most mutations accumulate as if no selection was happening at all. When a mutational effect is below the selection threshold, that mutation is 'effectively neutral'. It is unselectable. Its effect cannot rise above the genetic and environmental 'noise'. This is true regardless of whether it is beneficial or harmful to the organism. None of the realistic simulations resulted in fitness gain.

Nelson and Sanford addressed the question of whether or not selection could preserve biological information using an alternative simulation.[16] To do so, they rigorously compared results from Mendel experiments with results using one of the most popular evolutionary modelling programs out there, *Avida*. Interestingly, they discovered that an Avida--like model could only lead to evolutionary advance when extremely unrealistic parameter settings were employed in Mendel. *Avida* is highly unrealistic biologically. For example, *Avida* normally employs mutational effects that are grossly unrealistic (e.g. every step forward doubles total functionality/fitness). However, when realistic mutational effects were employed, there was no increase in fitness. Selection fails to create any new information, and any information already present degenerates toward 'zero', as shown by their detailed analysis of the inner workings of *Avida*.

Since the pioneering work of Kimura[17] and Ohno[18] in the late 1960s and early 1970s, evolutionary theory has recognized that, while most mutations are deleterious, individually their harmful effects are too subtle to be influenced by natural selection, and so cannot be effectively eliminated. Thus, a huge number of these 'nearly neutral' deleterious mutations accumulate continuously—like rust on a car. Even though an individual speck of rust is inconsequential to the function of an automobile, accumulating rust specks will eventually destroy it. Thus, mutations must be removed or the species will go extinct, and yet there does not appear to be any effective mechanism that can do this.

Two escape mechanisms have been proposed that, some have argued, might solve this problem. They are the 'mutation count' mechanism and 'synergistic epistasis'. These two mechanisms are similar. They aim to make the selection process more efficient, but in unrealistic ways. The mutation count mechanism works by (unrealistically) *counting* mutations in each individual, *ranking* the individuals by that count, then eliminating only those individuals above a certain count threshold. Nature does not operate that way.

Brewer *et al.* analyzed the mutation count mechanism.[19] They showed that it does not work, except when using extremely unrealistic parameter settings (such as forcing all mutations to have the same fitness effect). Whenever parameter settings were used that were even remotely

realistic, the mechanism failed completely.

Synergistic epistasis assumes an (unrealistic) fitness model for describing how multiple mutations *combine together* in their effect on fitness. The idea is that when mutations interact with one another, they might sometimes amplify each other's deleterious effects. Selection might then become more effective and the mutations could be removed simultaneously (essentially killing two or more birds with the same stone). This is different from the traditional view of epistasis. For example, when *many* genes work together to create a light-sensing cell in the retina, these genes have an effect greater than the sum of their individual effect. In other words, the whole eye is greater than the sum of its parts. Likewise, harmful mutations in these genes, *in combination,* have a greater harmful effect than the mere sum of their harmful effects (a few little changes

Figure 3. Mutation accumulation in the H1N1 influenza virus since 1918, from Carter 2019.[24] The published Brevig Mission strain from 1918 was used as the baseline for comparison with all available H1N1 genomes that had infected humans (dark diamonds) and swine (light diamonds) that were reported in the Influenza Research Database through late 2018. (1) The human H1N1 lineage goes extinct for the first time in 1957. (2) The human H1N1 reappears in 1976 after an accidental re-release in Moscow. This creates a disjunction in the trendline and allows for easy visual separation of the swine and human versions. Sporadic swine H1N1 infections occur in humans between 1976 and 2009. (3) The human H1N1 virus disappears from reporting in March 2009, the same year the swine H1N1 goes pandemic in humans. We first reported this in 2012. (4) Six years later, all currently circulating H1N1 viruses infecting humans derive from the swine virus. The second disappearance of the human version in 2009 occurred after 13% of the H1N1 genome had mutated due to the relentless effects of genetic entropy.

can have catastrophic downstream effects). That idea is well-established and is traditionally known as 'epistasis'. But 'synergistic epistasis' is quite different. It assumes it makes *no difference* whether the mutations are combined toward some useful combined function, such as sensing light. Instead it assumes any mutations, no matter what they affect, will have a magnified effect *in any arbitrary combination*. For example, it assumes harmful mutations to a toenail, the retina, and a tendon in the shoulder will have a magnified effect *in combination*, just as though they were doing something important together. This assumption is unrealistic. So unrealistic that evolutionary geneticists generally avoid it altogether (except when trying to solve such problems as genetic deterioration).

Baumgardner *et al.* examined synergistic epistasis.[20] They conclusively showed that synergistic epistasis does not help stop deleterious mutation accumulation. In fact, it greatly accelerates genetic degeneration and leads to rapid extinction.

Bringing it full circle

There was one other paper presented at the BINP meeting that is important for this discussion. Brewer *et al.* raised an interesting question in their paper on mutation accumulation in RNA viruses, which they modelled using Mendel.[21] Given the error rate inherent in RNA viral replication and the size
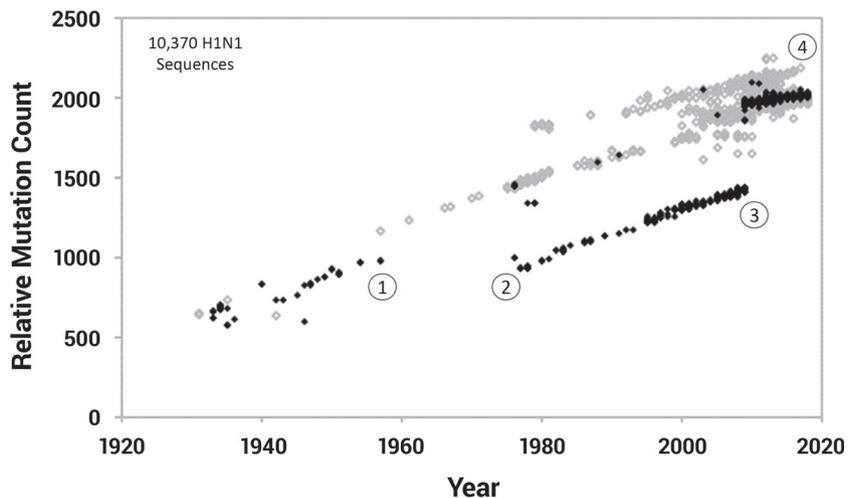
of the viral genome, RNA viruses are prime candidates for genetic entropy.

Carter and Sanford followed this up in 2012 with a study on the human H1N1 influenza virus, one of the most infamous RNA viruses in history, and the cause of the 1917/18 influenza pandemic that killed more people than died in WW1. We showed that mutation accumulation (despite lots of natural selection) led to the decline and eventual extinction of the virus (figure 3).[22–24]

This was the first time that genetic entropy theory was experimentally demonstrated in a biological system, completing the cycle from theory, to simulation, to application in the real world.

Ratcheting up the pressure

This flurry of papers from the Cornell symposium on biological information was not the end of the discoveries being made by Mendel. Rupe and Sanford wrote a fascinating paper that described a new principle they called 'Haldane's Ratchet'.[25] They set out to demonstrate that the famous theoretical problem known as 'Haldane's Dilemma' was indeed a serious challenge to evolutionary theory.[26] Haldane's Dilemma derives from theoretical work performed in the 1950s, where Haldane showed that, even when there are a sufficient number of beneficial mutations in a population,

it takes too much time for selection to amplify them to the point where all individuals carry the 'good' mutations. The amount of time involved is simply unrealistic.

Haldane estimated that it would take 300 generations for a beneficial substitution to become 'fixed' (as in 'stuck', the point where all individuals carried a given mutation) in most populations. Thus, in three million years (the then-assumed time required for ape-to-man evolution), a pre-human population could only 'fix' about 500 beneficial mutations. This is a trivial amount of genetic information, which means Haldane's Dilemma makes human evolution impossible. Rupe and Sanford used Mendel to simulate and experimentally validate that Haldane's Dilemma is very real. In addition, they showed that *net* fitness declined due to the accumulation of deleterious mutations while the beneficial mutations were going toward fixation. In other words, for every beneficial mutation that was selectively fixed, a large number of nearly neutral deleterious mutations simultaneously drifted to fixation. The result is that, even in the most generous scenarios, net fitness only went down—it never went up. This one-way process acts like a *ratchet*, hence the title Haldane's Ratchet, which is a hat-tip to another evolutionary paradox, Mueller's Ratchet,[27] that deals specifically with mutation accumulation in asexual species.

In 2015, Sanford and colleagues published on the 'waiting time problem'.[28] Evolutionists already knew that in all modest-sized populations there is a long waiting time before any specific point mutation will arise. This greatly amplifies the problem of Haldane's Dilemma, which assumes that all required beneficial mutations are already in the population.[29] But single point mutations do not generally create new functions. For decades it has been argued how many beneficial mutations are needed, and how much time is needed, to typically evolve a new function. In this paper, they used Mendel to model strings of mutations in a human-like population. They showed that even a string of just two mutations required roughly 84 million years to arise and become fixed in the population. Of course, the string will *appear* more often than that, but since most mutations are lost to drift,[25] even when beneficial, the real waiting time is much longer than the 'first appearance' time. A string of five point mutations would take approximately two billion years to arise, catch hold, and go to fixation! This problem is similar to Haldane's Ratchet—but more than a million-fold worse. The authors modelled different mutation rates, fitness benefits, and population sizes, but even when using extremely generous settings, the waiting time problem is devastating for neo-Darwinian theory, especially for species with long generation times (like humans).

Finally, Mendel was used to show that simulations that employed alternative population models created allele distributions that better matched the actual allele frequency distribution (as seen in the modern human population) than did simulations that employed evolutionary parameters
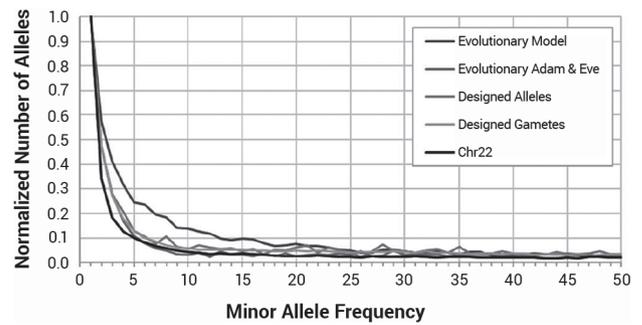

Figure 4. The allele frequency spectrum from several model runs compared to the real distribution, after Sanford *et al*. 2018.[30] This shows the allele frequency spectrum of all mutations found along chromosome 22 in the 1000 Genomes Project data (black line, to the left and bottom). Parallel to that are the results from several Mendel population models. Clustered near the 1000 Genomes data are an 'evolutionary Adam and Eve' model, in which a population of 10,000 individuals is allowed to mutate for tens of thousands of years before being reduced to a single pair, a 'designed alleles' model, in which the genomes of the biblical Adam and Eve are frontloaded with many neutral genetic variants, and a 'designed gametes' model, in which Adam and Eve's reproductive cells are frontloaded with a different genome in each cell. Standing off from the others is an evolutionary model that has no bottleneck and in which a population of 10,000 individuals is allowed to exist for many thousands of years. Clearly, the standard evolutionary model is the worst fit to the real-world data. (See original paper for a colour version of this figure.)

(figure 4).[30] These results are intriguing. Since Mendel has already been demonstrated to be behaving according to standard evolutionary theory, the fact that alternatives to standard evolutionary theory better fit the real-world data should make us question some of the assumptions behind the 'Out of Africa' theory, for example.

Success has brought criticism

A Google search for Mendel's Accountant will bring up multiple hits that criticize the program and the conclusions being drawn from its discoveries. Essentially none of these attacks are substantive, and many are highly misleading. It is clear that most people commenting on Mendel have not read either the documentation or the background papers. Thus, many evolutionists arguing against it don't seem to understand their own theory. This is probably due to the anonymous nature of the internet, and the level of expertise required to make comments online (i.e. no understanding required). However, one anti-creationist blogger (not trained in either biology or genetics, and it shows) tried to build a credible case against the genetic entropy thesis, and thus tangentially attacking Mendel's Accountant. John Sanford has rebutted that review in an important article on creation.com.[31] We are unaware of any peer-reviewed paper that attempts to refute the methods or conclusions of Mendel. After a decade of established work, there should be *something*. Their silence is telling.

Conclusion

Mendel's Accountant represents a milestone in our understanding of how the mutation/selection process operates. It shows what mutation/selection can and cannot do. For the first time, a robust computer program is able to successfully model realistic populations, track mutations through time, and rigorously test different evolutionary scenarios. No longer can evolutionists hide behind the mutation-count mechanism or the synergistic epistasis mechanism. No longer can they use selection as a 'magic wand' to wish away the theoretical, mathematical, or computational difficulties they face. Mendel is a powerful tool and should be used often. The collapse of the Darwinian mutation/selection mechanism is profound, and this news deserves more attention.

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