Bringing the Real World to Genesis: Why Evolution is an Idea that Won’t Die—IV [A Review]

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One mutation is found in hemoglobin, the oxygen-carrying protein within red blood cells. One of the molecules of human hemoglobin is beta-globin, and is composed of six genes; five are functional, and one right in the middle is broken. It is referred to as a pseudogene. This broken gene contains a series of errors that make it nonfunctional. This error is one that every human carries, and interestingly gorillas and chimpanzees also carry six beta-globin genes, and they are arranged in exactly the same way—five working copies surrounding a pseudogene. (Link)

What is interesting about this argument is that the eta-globin pseudogene, in particular, has long been known to be mutating at one-fifth the expected neutral mutation rate. Given that more and more “pseudogenes” and non-coding regions of DNA previously thought to be “junk DNA” are now being found to be functional to one degree or another, such a reduced mutation rate makes it very hard to definitively argue that the eta-globin sequence is a clear example of “shared mistakes” that have been passed on over the past 85 million years. It is also rather hard to imagine why or how a truly non-functional sequence would be maintained in the genome for such a long period of time without having been eliminated by random mutations (especially considering the cost required to maintain truly non-functional DNA).
And, ironically, just this year (January of 2013), a paper was published by Moleirinho, A., et al., demonstrating that the eta-globin pseudogene is in fact functional, playing a regulatory role and assisting in "gene switching" between fetal and adult forms of hemoglobin. Consider a portion of their argument as follows:

Several decades ago, a hypothesis was formulated holding an important regulatory role of HBD and HBBP1 in the Hb fetal-to-adult switch that matches quite well the assumption of strong negative selective forces acting on these sequences (Ottolenghi et al. 1979; Bank et al. 1980; Chang and Slightom 1984; Goodman et al. 1984). Over the past years, the β-globin cluster has been regarded as a complex genetic system and a paradigm of gene expression regulation. More recently, a boost of studies on the β-globin cluster have contributed to a better understanding of the mechanisms underlying the regulation of each gene in the cluster (Harju et al. 2002; Chakalova et al. 2005; Noordermeer and de Laat 2008; Sankaran et al. 2010). Remarkably, chromosome conformation (3C and 5C) analyses for the β-globin locus disclosed strong interactions between the LCR and the region encompassing both HBD and HBBP1 (Dostie et al. 2006; Sanyal et al. 2012). Furthermore, distinct spatial interactions of the LCR in fetal and adult stages were uncovered by another study based only in 3C assay in which HBD sequence was proposed to be enrolled in the maintenance of a transcriptionally competent structure at the adult stage (Beauchemin and Trudel 2009). These recent findings suggest that HBD and HBBP1 might be involved in chromatin looping in the human β-globin cluster, a crucial mechanism for temporal coordination of gene expression (Holwerda and De Laat 2012). Importantly, one SNP (rs10128556) in HBBP1 has been also identified as a modulator of HbF levels reinforcing the idea that this genomic region is indeed involved in the Hb fetal-to-adult switch (Galarneau et al. 2010).


So, I guess we just have to chalk up another one for the creationists don’t we? Yet another "pseudogene" bites the dust and isn’t so "pseudo" any more...

In any case, here are a few other interesting arguments from Long's article:

In DNA terms, Chimpanzees are the closest of the mammals to humans. The latest assessments have concluded their DNA is 98.8% matched to human DNA.

This is only true of protein-coding genes. This does not take into account the degree of differences that exist in functional non-coding genetic elements – like those that code for miRNAs (where ~8% of genetic sequences are unique to either humans or apes). Note also that when one is talking about protein-coding gene similarities, humans are 50% the same as bananas...
2. Most mammals have a gene that codes for an enzyme called gulonolactone oxidase, or GLO. This enzyme manufactures vitamin C, allowing mammals with this functional gene to not need any dietary intake of vitamin C. Humans, of course, need vitamin C in order to maintain good health, and interestingly human's have a remnant of the GLO gene that is broken. It has accumulated so many changes in its base sequence as to become nonfunctional. Assuming the viability of the evolutionary model, this very strongly suggests that every human has descended from a common ancestor that also had this broken gene. Yes, some primates—the ones we are most closely related to in terms of DNA patterns such as chimps and gorillas—also have a broken GLO gene. Other more distantly related primates do have a functioning GLO gene. As noted by Kenneth Miller, in the field of forensics, “this notion of unique, matching errors is widely used to determine when one document has been copied from another.” In the case of the GLO gene, the document we can analogize to would be the DNA code.

Now, it is interesting that among the many various substitution mutations in the “GLO” pseudogene that many, though not all, would be shared, to include a single deletion mutation that is shared by all primates (when compared to the rat of course). If not for common descent why would the sequences of human, chimpanzee, gorilla and orangutan reveal a single nucleotide deletion at position 97 in the coding region of Exon X? What are the odds that out of 165 base pairs the same one would be mutated in all these primates by random chance? Pretty slim – right? Is this not then overwhelming evidence of common evolutionary ancestry?

This would indeed seem to be the case at first approximation. However, in 2003, the same Japanese group published the complete sequence of the guinea pig GLO pseudogene, which is thought to have evolved independently, and compared it to that of humans [Inai et al, 2003]. Surprisingly, they reported many shared mutations (deletions and substitutions) present in both humans and guinea pigs. Remember now that humans and guinea pigs are thought to have diverged at the time of the common ancestor with rodents. Therefore, a mutational difference between a guinea pig and a rat should not be shared by humans with better than random odds. But, this was not what was observed. Many mutational differences were shared by humans, including the one at position 97. According to Inai et al, this indicated some form of non-random bias that was independent of common descent or evolutionary ancestry. The probability of the same substitutions in both humans and guinea pigs occurring at the observed number of positions was calculated, by Inai et al, to be $1.84 \times 10^{-12}$, consistent with mutational hotspots.

Some have noted that although the shared mutations may be the result of hotspots, there are many more mutational differences between humans and rats/guinea pigs as compared to apes. Therefore, regardless of hotspots, humans and apes are clearly more closely related than are humans and rats/guinea pigs.

The problem with this argument is that the rate at which mutations occur is related to the average generation time. Those creatures that have a shorter generation time have a correspondingly higher mutation rate over the same absolute period of time – like 100 years. Therefore, it is only to be expected that those creatures with relatively long generation times, like humans and apes, would have fewer mutational differences relative to each other over the same period of time relative to those creatures with much shorter generation times – like rats and guinea pigs.

What is interesting about many of these mutational losses is that they often share the same mutational changes. It is at least reasonably plausible then that the GULO mutation could also be the result of similar genetic instability that is shared by similar creatures (such as humans and the great apes).

This same sort of thing is seen to a fairly significant degree in the GULO region. Many of the same regional mutations are shared between humans and guinea pigs.
Why would both humans and guinea pigs share major deletions of exons I, V and VI as well as four stop codons if these mutations were truly random? In addition to this, a mutant group of Danish pigs have also been found to show a loss of GULO functionality. And, guess what, the key mutation in these pigs was a loss of a sizable portion of exon VIII. This loss also matches the loss of primate exon VIII. In addition, there is a frame shift in intron 8 which results in a loss of correct coding for exons 9-12. This also reflects a very similar loss in this region in primates (see Link). That’s quite a few key similarities that were clearly not the result of common ancestry for the GULO region. This seems to be very good evidence that many if not all of the mutations of the GULO region are indeed the result of similar genetic instabilities that are prone to similar mutations – especially in similar animals.

As an aside, many other genetic mutations that result in functional losses are known to commonly affect the same genetic loci in the same or similar manner outside of common descent. For example, achondroplasia is a spontaneous mutation in humans in about 85% of the cases. In humans achondroplasia is due to mutations in the FGFR2 gene. A remarkable observation on the FGFR2 gene is that the major part of the mutations are introduced at the same two spots (755 C->G and 755-757 CGC->TCT) independent of common descent. The short legs of the Dachshund are also due to the same mutation(s). The same allelic mutation has occurred in sheep as well.

3. Humans have 46 chromosomes—23 inherited from each parent. Apes, however, have 48, raising the significant question as to how humans and apes could possibly be related (particularly closely related) when humans are missing a couple of chromosomes. Well, this is where it gets interesting, because chromosomes have distinctive structural features with telomeres at the tips, and with a centromere at the center of the chromosome (see the graphic below). Quite unexpectedly humans have a fused chromosome #2. It has fused telomeres and two centromeres right where they would be expected to be if a fusion had occurred. Furthermore, genes on these two chromosomes are arranged in a pattern that is almost an exact match for corresponding genes on the two corresponding chimpanzee chromosomes. The match is so close that scientists have changed chimpanzee genes #12 & 13 to 2a and 2b so as to correspond to the human chromosome #2. This, of course, suggests an explanation for the “missing” human chromosome.

Despite the commonality of this argument in favor of common descent, I’m at a loss to understand what this particular argument is so convincing to so many people? Why is a chromosomal fusion event declared to be so “unexpected”? Consider that chromosomal fusions happen to be fairly common – even within the same species. In fact, there are humans alive today that have chromosomal fusions – and surprise surprise, they’re still human! – morphologically and functionally indistinguishable from other modern humans. Another example can be found with horses. Hybrids of the wild horse have 33 pairs while the domesticated horse has 32 chromosomal pairs. Also, domestic dogs and wolves of the genus canis have 78 chromosomes while foxes have a varied number from 38-78 chromosomes. Yet another example is the house mouse Mus Musculis, which has 40 chromosomes, while a population of mice form the Italian Alps was found to have only 22 chromosomes.

So, the different chromosomal numbers between humans and apes doesn’t necessarily indicate common ancestry. It is not evidence for when the event took place, nor is it evidence for the ancestry prior to that event. It could just as easily mean that similar creatures with independent ancestries originally had the same chromosome number and general banding patterns – a number that was later altered by fusion mutations in the human population during a population bottleneck. Given another dramatic population bottleneck in the future, such a transmissible fusion could easily happen again – in either apes or humans . . . or any other creature for that matter. That’s what’s clearly predictable here. Even those who believe in intelligent design (ID) understand that not all genetic features
require the input of intelligence. The simple fusion of two chromosomes, without any significant functional gain or loss, is easy to explain via random mindless processes and is actually fairly common. No big deal. Not very surprising or shocking – not even from an ID perspective. In fact, evolutionists would make exactly the same argument for the common ancestry of humans and apes without the fusion of chromosome 2. This fusion event really adds nothing to the argument. It simply presents no additional explanatory or predictive power to the argument for common descent beyond the simple observation that similarities suggest a common origin of some kind...

While it is quite reasonable that strong similarities, such as exist between humans and apes, do in fact indicate a common origin, that common origin is not necessarily based on common descent via slow genetic modifications selected by mindless nature over time from some shared common ancestor. Given the highly functionally complex differences between the two species which are being discovered more and more in recent years (especially in non-coding regions of the genome) it seems far more likely that the common origin of these differences, as well as the similarities, was based in deliberate highly intelligent design. The only event(s) that clearly do not require the input of high-level outside intelligence are events like random chromosomal fusions or other forms of random mutations which are very unlikely to produce any functional benefit beyond very low levels of functional complexity.

Well, what should we make of findings such as these? Are they all mere coincidences, or do they lead to the conclusion of common descent? The good news for traditional Adventist thinking is that none of these observations result in conclusions that are definitive on questions of common descent—just tantalizing data that seems to point that direction as determined by subject matter experts. For many this will be enough to casually dismiss this discussion and its implications. But this is not the end of the story, for we can be sure that there are many chapters yet to come. Yet, the data we have just discussed should cause us to pause before offering up knee-jerk responses of ridicule.

Shared similarities are admittedly rather easily explained via common descent. The real problems for the modern theory of evolution come when one tries to explain the functional differences between different gene pools beyond very low levels of functional complexity. Where things become completely untenable, to even a “ridiculous level”, for neo-Darwinists, is in trying to explain how qualitatively novel genetic systems could have evolved via the mindless mechanism of random mutations and natural selection beyond very low levels of functional complexity (i.e., beyond the level of systems that require at least 1000 specifically arranged amino acid residues to produce a given type of function). That’s the real problem for the ToE that these “shared mistakes” arguments simply don’t trump – not even close.

Those who have a defining narrative that would deny or ignore the data just discussed can easily find creationists who will be dismissive of the substantive points just made [thanks for your reference to my website here, but I hardly think my counterarguments are "dismissive" of the arguments presented for common descent]. So the problem for all truth-seeking laypersons is in whom to place trust for a study of very complex issues—the actual subject matter experts or the opinions of those who aren’t. The answer should be obvious.
Is this really a matter of trust or blind faith in this or that “expert”? Or, should the truth–seeking laypersons seek to learn and search out a personal understanding of the issues in play regarding such an important topic? Why put one’s faith blindly in the opinions of others who are clearly just as open and prone to bias and error as any of the rest of us?

Another point to consider—most credible experts will openly discuss both strengths and weaknesses of the findings they put forward. Those “experts” who misrepresent the known scientific reality by only presenting one-sided arguments as is done on many radio talk shows, where cherry picked data is presented and problematic data is avoided, are by their very approach untrustworthy. Those who openly discuss vulnerabilities are, by this measure, more credible. In the spirit of this latter point, I have provided readers in footnote form, a website sponsored by an Adventist who dismisses the findings we have just discussed. But should you review that material, please consider the general lack of any discussion of vulnerabilities of the arguments being made.

I’ve presented both sides of the topic in play in my own discussion of these issues on my website – along with references for readers to consider both sides. In contrast, you’ve only presented one side in print, with an off–barred reference to my website as a counter. However, you yourself did not present or discuss very many if any potential problems with the theories you’ve presented. Why not? How does this affect credibility?

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