2012 FOXP2 DNA Report

For 13 years we at the Starchild Project have known the Starchild Skull came from a being that was not entirely human, if human at all. First, it shares no physical characteristics with a normal human skull—none! Unfortunately, this astounding divergence in physical points of comparison never impressed mainstream scientists because they could, and often did, glibly explain all of them away by insisting: Nature can do anything! But that was never true.

Nature actually functions by strict rules that confine life to well-defined boundaries outlined by the unique genetic code of each species. No laws are more firmly established than the laws of genetics. Fifty eye-witnesses can say that a person committed a crime, but if DNA shows otherwise, the witnesses are ignored. DNA dominates in courts because it is the math of biology. It says what it says, again and again, with consistency you can stake your life on.

Within that life-and-death consistency, the human genome does contain slight variations. Tiny Pygmies and tall Watusis are black Africans with stark physical differences, yet both tribes are unmistakably human. Differences in their

genetic makeup make it impossible for two Pygmies to produce a Watusi, and vice-versa. Yet DNA can flex enough so that if one of each tribe were to mate, they could produce viable offspring, although the flexibility does not go beyond certain points. Something is either human, or it isn't. There is no in-between.

Because genetics is the math of biology, the Starchild's DNA provided the only means to overcome the mainstream explanation that it has to be a one-in-a-billion freak of Nature. Unfortunately, we had to wait nearly a decade while the technology for recovering and sequencing "ancient" DNA, such as the 900-year-old Starchild's, could be perfected.

Now such new technology has been in place for a few years, and its initial extraordinarily high cost has fallen within the reach of reasonable investment. Also, we now have enough partial analyses of the Starchild's DNA to know without doubt that when we can afford a complete inventory of its genome, it will prove to be radically different from humans.

This essay is designed to make the most crucial information about those partial analyses understandable to anyone. If you can take the 15 minutes needed to read it, you will learn about the three kinds of partial proofs we now have, what each one means, and why they will help the Starchild make history on a scale that seldom occurs in human lifetimes.

From New Scientist, Feb. 7, 2011 [text in brackets added]:

Fossils of the Denisovans, [a new extinct hominin and] close relative of Neanderthals, were discovered in Siberia in 2008. A draft genome was released in 2010 by Svante Pääbo of the Max Planck Institute for Evolutionary Anthropology in Leipzig, Germany, which revealed that Denisovans interbred with modern humans. However, each position in the genome was read only twice, so the fine detail was unreliable. The new genome covers each position 30 times over [so the fine detail is now highly reliable].

To understand the Starchild Skull's unique situation, the short paragraph above must be understood. In 2010, the Max Planck laboratory secured two "reads" (short for readings) through what are called "next generation" sequencing machines. That means they made the first announcement of a draft assembly of the Denisova genome with the average depth of coverage being around two. Each assembly includes thousands to millions of individual readings, and the depth of coverage indicates how many times each reading is repeated.

The draft data would have included gaps that remained in certain areas of coverage in the entire genome, so while the result would be highly indicative (the reason they announced it), the gaps would make it basically unreliable. This is always the case when data are obtained with "next generation" sequencing methodologies. However, conducting many reads closes nearly all

of the gaps, and ultimately provides a very high level of certainty for the results.

With the Starchild Skull, the partial results obtained by our geneticist at the DNA lab we work with are every bit as reliable, and as compelling, as those from Max Planck. He uses the same analytical techniques, and his results are what theirs are—partial but compelling. And, like the geneticists at Max Planck, to put our geneticist's results beyond all doubt, he has to complete them at least 30 times over, to the same extraordinary level of certainty.

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To understand what our geneticist has accomplished so far, let's start with nuclear DNA (nuDNA). It is found inside the nuclei of nearly all of the trillions of cells in human bodies (everything other than red blood cells and sex cells). It contains genetic material provided by our parents when our mother's egg is fertilized by our father's sperm. In humans, it produces a genome made up of over 3 billion base pairs comprised of what are called nucleotides.

In the illustration above, the four different nucleotides are distinguished by their chemical bases—Cytosine with Guanine, and Thymine with Adenine. Working together while always matched to each other, they form the base pair "steps" of the "ladder" that is DNA. The 3+ billion base pairs of the sprawling nuclear DNA genome contain hundreds of thousands of mutations called Single Nucleotide Polymorphisms, or SNPs. Among those hundreds of thousands, over 4,000 are known to be associated with genetic diseases or disorders.

In any cell's nucleus, most of its DNA is considered non-coding, non-functioning, or, as it is widely known, junk. As much as 95% of the nuDNA genome is considered junk because it doesn't produce any of the proteins our bodies need to survive. Our cells seem to do all of their normal housekeeping and functioning using only 5% of our nuclear DNA genome. However, 5% of 3 billion is 150 million, so apparently that is enough to accomplish the myriad biochemical tasks required to keep our bodies functioning as they should.

Nobody understands why our bodies need so much junk DNA. What we do know is that during every cell division, our bodies faithfully reproduce all of our nuDNA, including the junk, which means our bodies treat it as vital, so it must be considered highly conserved. (In this sense, the term "conserved" means "protected from change; not easily transformed.")

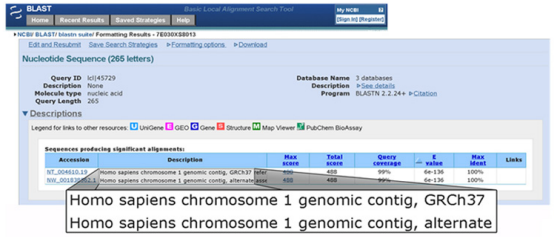
From the Starchild's nuDNA genome, our geneticist recovered and sequenced several dozen fragments that totaled well over 30,000 base pairs (bp). Though seemingly a large number, it is a woefully small percentage of the 3+ billion bp total (.001%). Nonetheless, those several dozen fragments tell a compelling story about the Starchild Skull's DNA.

Our geneticist sent those unknown genetic sequences to be compared with millions of sequences contained in the massive database at the National Institutes of Health (NIH), in Maryland. That database is extremely

comprehensive, covering the accumulated genomes of thousands of different organisms—from mammals to reptiles to crustaceans to bacteria.

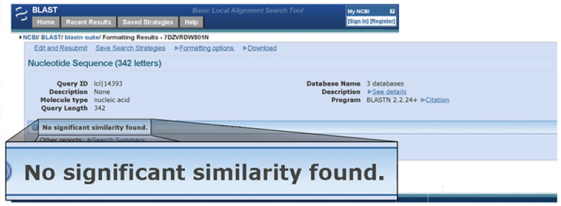
When comparing the Starchild's sequences, the search parameter ranged from an exact match of the entire base pair string, to matches that were similar to any segment of any fragment. Using these exceptionally broad criteria, many Starchild fragments could be matched to genetic sequences in the NIH database. Some of those were comparable to human sequences, which meant they were human-like, though not necessarily human.

The human genome has large numbers of corollaries in the world around us. Humans share 97% of our genes with chimps, 95% with gorillas, 70% with rats, 65% with mice, and 26% with yeast! Thus, nearly everything on Earth is, in some way, genetically interrelated with humans, so it is not unusual that some of the Starchild's nuDNA is found to be human-like.



What is unusual, and shockingly so, is that there are segments of many other fragments of the Starchild's nuDNA for which no close matches could be found in the NIH database!

This is not unheard of, nor impossible, but it is a significant indicator that something about the Starchild is not entirely human. It strongly suggests that some aspects of the Starchild's DNA might not be found on Earth at all! Again, this is not absolute proof. We need many additional readings through modern sequencing machines to confirm it. However, we take this initial partial result as a strong indication that the Starchild is not entirely human.



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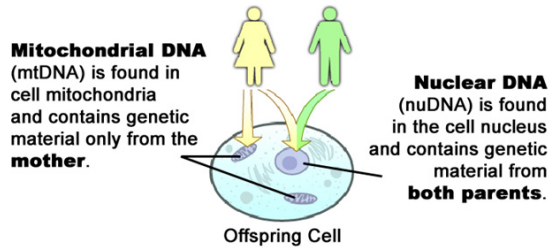
An even stronger indication of this came later, when four fragments were recovered from the Starchild's mitochondrial DNA (mtDNA), which is found in tiny parcels floating like raisins in pudding within the cytoplasm between any cell's nucleus and its outer wall. MtDNA is passed along to all generations through the female line, in her much larger egg. Males do not contribute to mtDNA because sperm contain only the male's genomic package, nothing else.

In sharp contrast, the mtDNA genome is vastly smaller and much more tightly packed, and it contains a very specific number of base pairs: 16,569, compared to the 3+ billion in nuDNA.



All DNA is equally susceptible to mutations, which are malformations or irregularities in the arrangement of coding regions that prevent them from functioning properly. Mutations can occur at any time and at any point in

nuDNA or mtDNA. The large amount found in nuDNA (hundreds of thousands of SNPs) is there because so little of it (5%) actually works to keep our bodies running. Mutations can steadily accumulate in the 95% called junk because their impact in non-coding regions seldom causes adverse impacts for those who develop them, and thus they can be—and in most cases are—passed on to succeeding generations.

In mtDNA, the exact opposite is true. Nearly all of mtDNA (98% or more) functions at a very high level of efficiency. Thus, if a mutation occurs in the working part, there is a very high likelihood its impact will be averse to the point of causing death, and therefore will not be passed to subsequent generations. In short, mutations in mtDNA tend to be eliminated from the population by the death of the individual in whom it occurs. This makes the DNA in mitochondria extremely highly conserved, which means it changes very little over time.

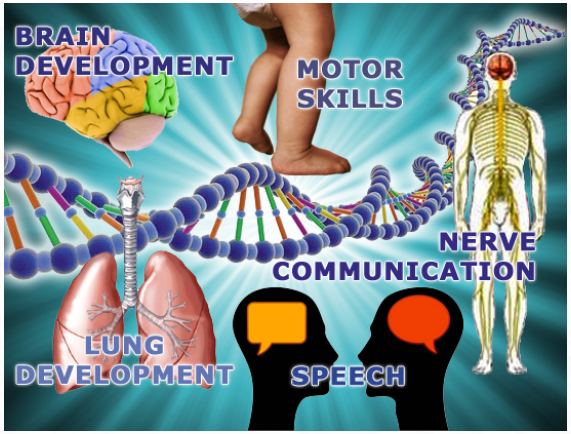
Among the 16,569 base pairs in each mtDNA genome, a maximum of only 120 vary between all humans. The 120 maximum is found in descendants of the first humans, who originated in southern Africa around 200,000 years ago. The rest of us carry fewer mutations because our ancestors did not develop until well after the first humans appeared.

No matter how many I carry, or you carry, all are found in the 2% of mtDNA that is not absolutely essential to the processes of our lives that they manage. This is why so few variations exist in mtDNA. Though physically quite small, the proper functioning of the mitochondria is essential to life, so permanent mutations must occur in nonessential areas.

In terms of this all-important mtDNA in the Starchild, our geneticist has recovered four reasonably large fragments which together total 1,583 base pairs, or 9.55% of the 16,569 base pair total for humans. As before, this is only a partial result, but also as before, it is highly indicative of what the final result of a full mtDNA genome analysis will be.

Within those 1,583 base pairs, the Starchild carries a grand total of 93 variations that are different from the extremely highly conserved human mtDNA genome. That is 93 in only 9.5% of the genome! It's already near to the maximum of 120 variations in human mtDNA. If we do a simple but highly reliable mathematical extrapolation, expanding the 9.5% out to 100% (times 10.5) we find that 93 established variations extrapolates out to 977 variations!

Remember, the maximum of variations in human mtDNA is 120. Neanderthals carry 200. The new hominins, Denisovans, carry 385. The Starchild extrapolates to 977! However, we must be clear about what that 977 means. During the course of repeated mtDNA sequencing, a high probability exists that several of the variations found will not hold up as valid. Some are likely to be established as errors. Because of that likelihood, let's be overly conservative and err well on the side of caution. Let's say the Starchild's mtDNA will fall in the range of 800 to 1000 variations. Compare that range to the human 120. What does it mean?



Based on this partial mtDNA recovery result, which must be repeated many times before it can be considered fully reliable, the Starchild Skull is not from a human being. We will no doubt hear arguments from mainstream scientists insisting it is some new kind of humanoid being, but it would have to be an exceptionally variant humanoid, something far away from Neanderthals and Denisovans, something nearly as genetically different from humans as chimps, which have 1,500 of those mtDNA variations compared to our 120 maximum.

To resolve the debate that will swirl around the Starchild's genetic status, its full genome will have to be compared with the human genome, the Neanderthal genome, the Denisovan genome, and, for good measure, chimps and gorillas, and other higher primates. Ultimately, a determination will be made, for good or ill, like it or not, no matter how inconvenient the result might be. But before we get to that point, we have to meet a new player in the game.

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Our geneticist has now recovered a fragment of the Starchild's DNA that is so powerfully convincing, even standing alone, we are confident it provides a tipping point in our quest to recover the Starchild's entire genome. He has secured a fragment of a gene from the 5% of human nuclear DNA that codes for proteins, and it does most of the work of keeping our bodies functioning as they should. This gene is not only functional, it is a highly functional "master gene," one of the most vitally important genes in the body of any species on Earth.

Virtually any complex species has a variation of this gene, and it is without question one of the most highly conserved genes in the human body. It is the FOXP2 gene. That odd name comes from its technical title: Forkhead Box P2, or FOXP2. Here is one of a wide variety of illustrations that try to capture its vast importance in a single image.

In any creature, the overwhelming importance of their FOXP2 gene is that it controls a "downstream" cascade of genetic processes in hundreds of other genes, all coordinating the formation of various parts of a body as it gestates and grows to maturity. In mammals and other "higher" species, any single flaw in FOXP2, any isolated mutation or variation, can cause a severe negative impact in some of the most important aspects of development: the function of the brain, the sound or speech mechanisms, the lungs, heart, guts, and nerves, among others. Because it is so utterly vital, it is even more highly conserved than mtDNA.

Recall that in the 16,569 base pairs found in the mtDNA genome of normal humans, as many as 120 variations can be found in the first of us, southern Africans. That percentage of difference is quite small, only 0.7%. Compare that with the FOXP2 gene, which in normal humans is 2,594 base pairs long, and contains no variations. 0%! None! Nada! Every normal human has the exact same array of FOXP2 base pairs as every other normal human.

This is not to say mutations never occur in FOXP2. They can and do, and a number of them have been found. However, every mutation is debilitating in some way, and because FOXP2 is vitally important to so many bodily functions, most mutations in it will cause termination of life. When termination does not occur, the mutation's impact on its host is usually severe.

In one well-studied mutation in the section of the gene that influences speech development mechanisms in humans, those who inherit it will never be able to speak. This has led some to suggest FOXP2 is a language gene, or a speech gene, but that is not the case. Speech is much too complex an arrangement of working parts to be so simply controlled, although a properly functioning FOXP2 gene is an essential part of the speech-development equation.

The key point to understand is that while a tiny amount of survivable mutations are possible in FOXP2, every one that occurs presents debilitating or life-threatening consequences, so to this point in time none have been passed on to the general population of humans. Therefore, in the vast, vast majority of humans, the FOXP2 master gene is absolutely identical.

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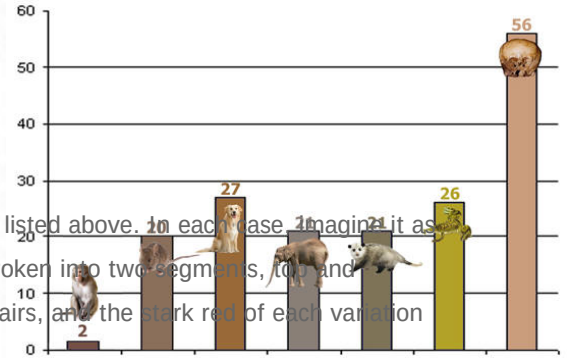
With that said, let's examine the fragment of Starchild Skull FOXP2 sequenced by our geneticist. Of the entire 2,594 base pairs of the normal FOXP2 gene, our fragment is 211 base pairs that come from a segment near the center of the gene. If the same 211 base pair section were isolated from any normal human, every base pair would be exactly the same as what is found in any other human. There would be no difference in any of them.

Okay, ready....brace yourselves. The Starchild's 211 base pair FOXP2 fragment has a grand total of 56 variations! Now, while extrapolating this 211 base pair fragment is a bit more of a stretch than extrapolating the four combined fragments of mtDNA we discussed earlier, doing so does provide something to think about. Divide 2,954 by 211, and you get 12.3. Multiply 12.3 by 56, and the range of total variations in the Starchild's FOXP2 base pairs would be 600 to 700! So let's be crazy conservative and say it's only 200 or 300. It is still astounding in a super highly conserved gene that in normal humans has no variations at all!

If we compare the same section from a rhesus monkey's FOXP2, only 2 of its 211 base pairs would vary from any human. If it were a mouse, it would be 20. If a dog, 27. An elephant, 21. An opossum, 21. A Xenopus (a kind of frog), 26. So dogs and frogs are the most different, at 27 and 26 base pairs respectively.

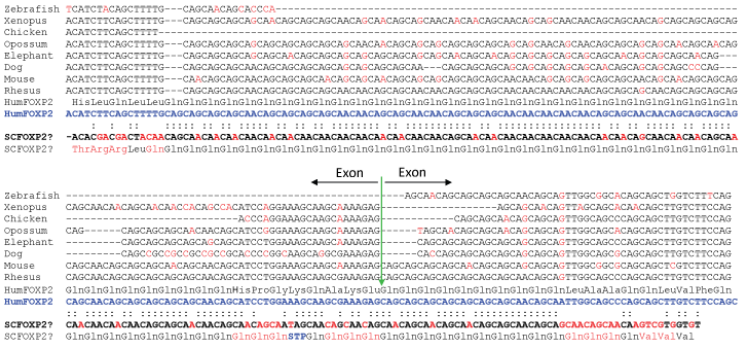
To put this in perspective, let's imagine that when alive, the Starchild was indeed some unknown humanoid. No matter how different from humans it might have been, to be in the humanoid family its FOXP2 gene would have to be in the range of 1 or 2 or at most 3 base pair variations from a normal

human. To go past 5 or 10 would put it into another class of species. 20 to 25 would put it in the range of mice and elephants, and dogs and frogs. To have 56 is to put it in another realm, another dimension entirely. It is utterly unique.



To verify this radical statement, below is the actual comparison of the Starchild's FOXP2 fragment with the same gene segments of some of the species listed above. In each case, imagine it as a string of 211 base pair nucleotides, although to fit into this format it must be broken into two segments, top and bottom. Notice the steady blue of the human nucleotides that make up its base pairs, and the stark red of each variation in the other species.

Notice the SCFOXP2 line directly above, beginning with "Gln." It lists the amino acids determined by three-letter codons. Codons establish rules by which information carried by genes is translated into proteins, which are molecules that perform various functions in cells and eventually in bodies. These groupings, known as triplets, are formed from the single-letter nucleotides—C, A, G, T—and they represent a blueprint for building the proteins that are encoded by each gene, using the standard set of 20 amino acids that our bodies utilize.



The fragment shown above encodes a peptide (a stretch of amino acids) that is very unusual in the human FOXP2 gene. It contains a long stretch of 40 triplets, each coding for the same one of the 20 amino acids—glutamine (Gln), encoded by either a CAA or CAG triplet. After the stretch of 40 Gln in a row, there is a shorter stretch with six other triplets and one more Gln, then another stretch of 11 Gln in a row. In the Starchild FOXP2 fragment, there is also the 40 Gln tract, plus the 11, but it also contains several more glutamines.

In the 211 base pair fragment from the FOXP2 gene in normal humans, no variations occur among the amino acid sequence in the FOXP2 protein, and the coding pattern for Gln (using either CAA or CAG) is exactly the same not only in humans, but essentially in all primates. (Compare only 2 amino acid variations in a rhesus monkey, which is not even a great ape.)

In the Starchild Skull, we find 16 amino acid variations in this fragment, which despite all those differences unmistakably resembles the human FOXP2. Yet it demonstrates a coding pattern that is wildly different from all species shown above. This is an astounding contrast!

Notice, too, the Starchild's 40 Gln stretch continues for 3 more, making it a 43 Gln stretch, where it reaches what is known as a "stop" codon, TAG, signified by the blue all-cap STP near the middle of the bottom line. The presence of

this "stop" codon inside the protein is quite strange, since it will result in production of an abnormally shortened FOXP2 protein devoid of the downstream regions that are critical for important functions of this protein.

It is always possible that some kind of sequencing error has been made, so it needs to be repeated to confirm this initial analysis. In any case, after the mysterious "stop" codon, a new Gln stretch begins and continues to only three amino acids from the fragment's end. This, too, is wildly different from the human sequence, but as with the other anomalies, further research is needed to determine what altered functions these differences cause.

Another comparison to make is to remove the Gln stretches from different species and examine what is left. For example, if we analyze the entire FOXP2 gene in humans and chimps, our closest genetic relative, with the Gln stretches removed from consideration, then only 2 amino acids (depicted by the three-letter codons) are different. The same 2 are found in gorillas and other higher primates. In mice, the difference is 3 amino acids.

If we remove the Gln stretches from the Starchild's fragment of FOXP2, only 7 amino acids remain to be compared to the corresponding amino acids of the human FOXP2. These are the first four, at the beginning of the fragment, and the last three, the end of the fragment, and all 7 amino acids are different! Whatever we might say about this comparison, it is certainly not between two humans, or anything near two humans.

In addition to having a "stop" codon in its last quarter, the Starchild fragment is also missing the large intron (marked with a vertical green arrow) that normally intervenes in the human gene and in the gene of other species. This suggests that the Starchild fragment could be a pseudogene, dysfunctional ancestors of normal genes that have lost the ability to encode proteins, or are otherwise no longer capable of being expressed in a cell. This means they are nonfunctional, and are therefore another form of junk DNA.

Suggesting the Starchild's FOXP2 fragment might be a pseudogene immediately collides with the fact that there is no currently known human FOXP2 pseudogene. Because it is a master gene, it must always function properly, and if it doesn't function properly in even a small way, very negative things happen to the individual carrying the variation. Thus, since a human FOXP2 pseudogene is not known to exist, if it turned out that the Starchild Skull carried one, that would clearly establish it as not human.

What's the bottom line? That can only be determined when the entire Starchild genome is recovered and compared —nucleotide by nucleotide, base pair by base pair, codon by codon, amino acid by amino acid—with humans, Neanderthals, Denisovans, chimps, and gorillas. Whatever it is, most of the preliminary evidence indicates it is quite distinct from humans.

Most important, perhaps, to keep in mind is that our FOXP2 results are preliminary, as are the results from the earlier nuclear DNA fragments, and the mitochondrial DNA fragments. All three preliminary results are highly indicative

of what the final result will be, but they cannot be considered absolute proof. They can, however, be considered proof that absolute proof will come when the Starchild's entire genome can finally be recovered.

DNA Testing

- 2012
  - **Fragment of the Starchild's FOXP2 Gene is Recovered**
  - 2012 Starchild Skull Genetics Abstract
- 2011
  - Scientific DNA Analysis Report
  - The DNA Report in Laymens Terms
- 2010
  - Preliminary Nuclear DNA Findings
- 2003
  - Trace Genetics DNA Report

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51 comments

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  Garry Nolan · Top Commenter · Stanford University

  I have previously analyzed the sequence that was on the website and I see scant evidence that this is FOXP2. It is a glycine rich region where even the third base codons are often different from that found in mammalian FOXP2 genes or primate relatives. Many proteins have GLN rich regions. I made this point to the StarChild team several months ago. It is deceiving to continue claim this is FOXP2 in my opinion. Gregor Stewart below notes that this is "just" a GLN rich region (also implying the well known fact that poly-GLN tracts are not uncommon). Indeed, if you look at the flanking non-GLN region you see there is zero homology at the flanking regions, further suggesting this is from another gene. The claim that this is from FOXP2 is only detracting from your overall goals. Until you have the flanking DNA and non-GLN sequence from FOXP2 it's very easy to conclude the sequence could be from contaminating DNA or another gene.

  I have also looked at the other sequences provided me by Lloyd earlier in 2012 and as far as I can tell what I was provided looked far more like metabolite genes from bacteria and not "mitochondrial" as was proposed. This contamination possibly occurred during the "washing" the skull underwent after it was initially buried by the original discoverer (according to Lloyd) and then washed downstream post a rainfall/deluge (again as related to me personally by Lloyd). Plenty of opportunity to get contaminated if you are washed out -- from being buried in the ground near a stream -- and then found downstream and dug up from the mud. Possibly the skull was washed at other times-- giving plenty of time for bacteria to grow on remaining organic materials from the skull. This would provide a treasure trove of contaminating DNA. Maybe there is new evidence, or the team has used sequence specific primers to amplify "known" primate genes, but one should worry about contamination (even from material "deep" in the bone) given how we know the skull was handled over the years.

  Lloyd kindly brought the skull to Stanford and we did 3D tomography together on the skull with the most advanced machine we had on campus at the time in the Medical Center (2012). Lloyd was provided the 3D imaging data. No evidence of the fibers could be found-- though that result could be inconclusive in that the fiber size might have been below the resolution of the device or the fibers could otherwise be transparent to the instrument we used.

  I have previously suggested to the team they should be careful of the DNA results given the potential for contamination issues. My suggestion has been to use PROTEIN SEQUENCE from the bone or residual tissue. If the skull is considerably different in terms of evolution this might be represented in protein sequence of proteins in bone

material. If one is going to believe the logic (some would like to think) that the FOXP2 gene has changed so drastically as to alter the third position codon across several residues in the polyglycine tract as compared to that in mammalian homologs (and has apparently altered the flanking protein sequence as well and created new intron boundaries as claimed) then it's easy to believe that significant evolutionary drift or adaptation might be similarly found in the collagen sequences of the skull (or other proteins that are found in the viscera or tissues of the skull). There are several grams of protein likely to be available in the skull (collagen and other expected bone proteins?) that cannot be "contaminated" by bacteria since bacteria and fungi do not have collagen and bone. The team should strongly consider sequencing these proteins directly and not only trying to look at the DNA.

I am not trying to debunk the skull's origin or the overall goals of the project. The skull is interesting or I would not have taken the time to do the 3D imaging here at Stanford. I am saying the data you have so far are insufficient to claim FOXP2. Not even close to claiming such... and I suggest there are possibly better ways to get at the skull's provenance than the DNA, given that contamination can always be claimed against you. Protein information can be as valuable as the DNA. And you have a lot of protein in that skull with which to work I suspect and it's harder to claim contamination of the proteins if you look at the right ones in privileged/protected places in the specimen.

Reply · 19 · January 1 at 1:44pm