**A Tale of Two Chromosomes**

by Jean Lightner on November 14, 2007

**Abstract**

*There are many anatomical similarities between humans and apes. Our chromosomes are similar as well. But do human chromosomes hint of chimp ancestry?*

Evolutionists can be excellent storytellers. For example, Dr. Ken Miller, a biology professor from Brown University who testified against Intelligent Design (ID) at the Dover trial,[[1]](#footnote-1) tells an engaging story that he claims is compelling evidence for evolution. The problem is that because of his naturalistic assumptions, he himself is unable to distinguish fact from fiction, science from conjecture.

**Background**

Humans normally have 46 chromosomes. However, sometimes two chromosomes will fuse together to form one big chromosome. Centric fusions are where two acrocentric chromosomes (chromosomes with the centromere very close to one end) fuse to make a large metacentric chromosome (one with the centromere near the middle). It is estimated that around 1/1000 people carry this type of chromosomal rearrangement. While they are sometimes associated with problems such as infertility or serious chromosomal aberrations in the offspring, often they are asymptomatic.[[2]](#footnote-2) This is because all of the necessary information is there in the proper amount; it is just packaged differently.

**The tale of missing chromosomes**

There are many anatomical similarities between humans and apes. Our chromosomes are similar as well. We can see these similarities in the banding patterns of the chromosomes. One obvious difference between the human and ape karyotype is that apes have 48 chromosomes (24 pairs) and humans normally have 46 (23 pairs). Dr. Miller likes to tell an entertaining “who done it” type story asking where the missing chromosome pair went. He then points out the scientific evidence for a fusion event on human chromosome 2. There is evidence that implies a fusion event may have occurred.[[3]](#footnote-3) Human chromosome 2 corresponds to ape chromosomes 12 and 13. Dr. Miller states, “Our chromosome number 2 was formed by the fusion of two primate chromosomes.”[[4]](#footnote-4) Dr. Miller *assumes* common ancestry and the number of chromosomes is consistent with his belief. However, he misses other important evidence that contradicts his basic claim.

Most importantly, reliable eyewitness testimony is more powerful than circumstantial evidence in establishing historical details. The Bible, inspired by the Creator himself, indicates that humans were created in the image of God and distinct from other animals.[[5]](#footnote-5) Humans are clearly distinct from other animals in cognitive and language ability. Occasionally, the ability of chimps to use tools or simple sign language is touted as evidence for their close relationship with us. In reality, chimps are not significantly different in these areas from many other mammals and birds (except that they can use their hands more like us). Chimps lack the anatomy for human speech. Ironically, a few birds have been known to use human language quite well, at least for an animal.[[6]](#footnote-6) Simple tool making ability is also seen in a variety of animals.[[7]](#footnote-7) While intelligence in animals is quite fascinating, it is still significantly different from that of humans and gives no hint of common ancestry. The similarities are much more easily explained by the fact that these animals all had a common designer who reused certain excellent design elements much like engineers do in their creations today.

**Observed patterns of chromosomal rearrangement**

Dr. Miller’s enthusiasm about this chromosomal rearrangement may be tied to the older notion that such mutations are the basis for speciation.[[8]](#footnote-8) This belief was shown to be overly simplistic decades ago when papers appeared describing chromosomal variations which were not eliminated by selection. One intriguing example is a single species of rodent (*Holochilus brasiliensis*) where 26 different karyotypes were identified in the 42 individuals tested.[[9]](#footnote-9) Chromosomal rearrangements have been identified within many ruminant species. There are examples in both goats and sheep where individuals with one or more centric fusions are phenotypically indistinguishable from other animals.[[10]](#footnote-10) One researcher who studied sheep carrying up to three different centric fusions concluded, “It is now considered that there is little or no evidence to suggest that centric fusions in a variety of combinations affect the total productive fitness of domestic sheep.”[[11]](#footnote-11) So, the bottom line is that centric fusions themselves do not inevitably result in a new species. It is conceivable that some apes exist with 46 chromosomes. Yet these animals will be distinctly apes; they will not be “evolving” to become a human. If the observed evidence is really from a fusion, it is best explained by the fusion of two *human* chromosomes.

**A diversion from the real issue**

The biggest problem with Dr. Miller’s story is that it distracts the audience from the real issue. It is not the number of chromosomes that is really a significant difference between humans and apes, but the *information* contained on those chromosomes. According to the evolutionary scenario, our apelike ancestors underwent major anatomical restructuring to develop upright posture, speech ability, and an astounding increase in cognitive function all by random, chance processes. Such profound changes were never observed; they are inferred because evolution has an atheistic basis and assumes there is no creator.

Despite the superficial similarities between human and ape chromosomes, there are important differences on the molecular level. There are many protein coding genes in humans that are distinctly human and are not found in chimps. Perhaps more significantly are the differences in genes that don’t code for proteins. Genes have been described which code for microRNA (miRNA). The miRNA molecule is not translated, but acts directly to control gene expression. A single miRNA can regulate the expression of dozens or even hundreds of genes. A study of miRNAs expressed in the brain found 51 of 447 new miRNAs were distinctly human and 25 were only found in the chimp.[[12]](#footnote-12) The idea that so many genes were altered so that they are expressed in the proper concentration according to cell type and can effectively control the many different genes they regulate is not what we would expect of chance processes.[[13]](#footnote-13) It is more rational to believe that God created humans distinct from chimps, just as He tells us in the Bible.

**Blind to alternatives**

While the evidence for a fusion appears consistent with the evolution model, Dr. Miller implies that it is inconsistent with ID or creation models. He makes the ludicrous claim that the only way creationists can respond to this evidence is: “That’s the way the designer made it.”4 This statement reveals Dr. Miller’s inability to think outside his paradigm. As a creationist who finds chromosomal rearrangements fascinating, I can honestly say I never thought of that possibility. One possibility I had considered is that humans and apes (and perhaps other animals too) were created with the same number of chromosomes with similar banding patterns.[[14]](#footnote-14) Since chromosome numbers vary within created kinds, it is not in the chromosome number where we should expect the most significant differences to lie, but in the coded information.

Although Ken Miller’s story does not properly consider current scientific understanding of chromosomal fusions or significant genomic differences between apes and humans, he promotes it enthusiastically to support his belief that humans descended from apes. Furthermore, he is ardently opposed to teaching intelligent design in the schools, claiming that it is not scientific.[[15]](#footnote-15) He appears to be blind to the fact that the belief that humans descended from apes is a religious (atheistic) one; such changes have never been observed. Thus, he is not able to distinguish between science and religious indoctrination.

**True science and the Bible believer**

Despite the misunderstanding and wild story telling of evolutionists, science is truly a fascinating and rewarding field for Christians who believe the Bible. The sciences were founded by people with a strong Christian worldview.[[16]](#footnote-16) There are still many fascinating questions waiting to be answered. For example, why do chromosomal rearrangements occur? It has been pointed out in the literature that they are non-random.[[17]](#footnote-17) Do they have a purpose? (Evolutionists aren’t supposed to ask this.) Do they play a role in speciation? If so, how? Do they help animals adapt to new environments? Why are there times when they cause problems (i.e. some carriers have a high percentage of unbalanced gametes[[18]](#footnote-18) which results in infertility or abnormalities in their offspring)? How can they become fixed in a population? God’s world is out there waiting to be explored. The truth is far more fascinating than fairy tales.

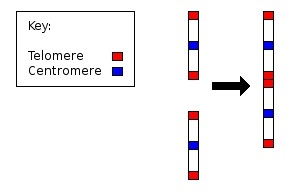
**Chromosome Tales and the Importance of a Biblical Worldview**

by Jean Lightner on June 18, 2014

**Abstract**

*Evidence never speaks for itself; it must be interpreted. When it is interpreted in a particular worldview, it can sound very convincing that the evidence supports that worldview. This was the case for the proposed chromosomal fusion that supposedly resulted in human chromosome 2. It was promoted as unequivocal evidence that humans and apes shared a common ancestor. In a biblical worldview, it is possible for a chromosome to have resulted from the fusion of two smaller chromosomes. However, there were details about the story that didn’t make sense. The biblical worldview provided the motivation to dig deeper. Further investigation now makes it clear that human chromosome 2 was not derived from a fusion of ape chromosomes; its structure is consistent with being designed by a wise Creator.*

In my lifetime I have seen a number of supposedly powerful arguments for evolution come and go.  Generally, they seem powerful because it is implied there is only one way to interpret the evidence, and only an evolutionary interpretation is given.  I have found that the biblical worldview is far more robust, and it is only a matter of time and some research before it is clear that the evidence is better explained by a biblical model.

Human chromosome 2 was said to have been formed by the fusion of two primate chromosomes that remain separate in chimps. It was supposed to be an end-to-end (telomere-to-telomere) fusion. Known fusions in mammals are different in that they occur near at least one centromere region.

A few years ago I wrote about one example of a “compelling” evolutionary argument, the supposed evidence for a fusion involving human chromosome 2.[[19]](#footnote-19)  According to Dr. Ken Miller, this was incredibly powerful evidence of common ancestry between humans and apes.[[20]](#footnote-20)  Since apes have 48 chromosomes (24 pairs) and humans have 46 (23 pairs), evolutionists propose that a fusion occurred to account for the difference.

In a YouTube video Dr. Miller showcases the supposed fusion as a prediction of evolution and boldly states:

What must have happened is that one pair of chromosomes must have gotten fused. So we should be able to look at our genome and discover that one of our chromosomes resulted from the fusion of two primate chromosomes. So we should be able to look around our genome, and you know what? If we don’t find it, evolution is wrong - we don’t share a common ancestor.[[21]](#footnote-21)

Chromosome fusions do occur.  The topic is of particular interest to me, so in my [previous article](https://answersingenesis.org/genetics/dna-similarities/a-tale-of-two-chromosomes/) I highlighted a number of assumptions hidden in Dr. Miller’s argument.  Evidence of a fusion would not be an automatic “slam dunk” against biblical creation.  Interestingly, since that article was written, there has been considerable research done suggesting that there never was a fusion on human chromosome 2.  According to the quote by Dr. Miller above, this would be a “slam dunk” against evolution.

**What Is Really in the Scientific Literature?**

First, an investigation into the scientific literature revealed that scientists didn’t find the proposed fusion location by hunting for sequences that appeared to be a fusion site, as Dr. Miller had suggested.[[22]](#footnote-22)  The idea of a fusion on chromosome 2 was suggested based on the assumption of common ancestry and  some similarities between chimp chromosomes 12 and 13 (now 2A and 2B) and human chromosome 2.[[23]](#footnote-23)  The idea of a fusion is certainly a reasonable proposal within the evolutionary model.

Secondly, the proposed fusion site did not look like those documented in other mammals.[[24]](#footnote-24)  Fusions known to occur in animals are characterized by sequences known as satellite DNA, and most often occur around centromeres.  The proposed fusion on human chromosome 2 was supposedly a telomere-to-telomere fusion, lacking satellite DNA.

Further, the proposed fusion site did not look like evolutionists expected.  It should have contained long stretches of a tandemly repeated sequence (TTAGGG), but this pattern was not seen.  Instead this sequence was not as prevalent as expected, and often it was not repeated in tandem (i.e. two or more in a row).  This surprised evolutionists, prompting the question “why are the arrays at the fusion site so degenerate?”[[25]](#footnote-25)

There was an additional surprise.  The proposed fusion site was surrounded by unexpected features.  For over 600,000 bases surrounding it, there exist many different types of human-specific genes – none of these genes are found in the chimp.  Telomeres do not contain genes.

In addition to the fusion site, there is supposedly the “remains” of a second centromere.  These were identified based on a type of alphoid sequence that is commonly found throughout the human genome in many locations, so this sequence is not specific to centromeres.  Thus, it was not too surprising they were able to find this sequence close to where the “fossil centromere” was supposed to be.22

All this shows that lay-level evolutionary arguments do not necessarily represent what is known from the scientific literature.  In fact, in my experience, the more compelling the evolutionary story, the less it resembles reality.  Evidence doesn’t speak for itself; it must be interpreted.  If someone presents evidence that appears to contradict the biblical history, one excellent response is to investigate more fully to see what is going on.  In fact, that is what has been done.

**Investigating Further**

Further research by creation scientists reveals even more compelling evidence against the idea that human chromosome 2 was formed by a fusion, much less a fusion of ape chromosomes.  A closer look at the supposed “fossil centromere” has shown that it is not in the correct location assuming it originated from such a fusion.[[26]](#footnote-26)  Further, the alphoid sequence in   humans (including at the alleged fossil centromere) does not match the alphoid sequence found in the chimpanzee.26

Focusing on the supposed fusion site itself reveals even more intriguing information.  Using the BLASTN tool, the alleged fusion site aligned (due to nucleotide similarity of 80% or more) with many regions on human chromosomes and only a few on chimpanzee chromosomes.  However, it did not align with the portions of the chimp genome that were supposedly involved with the fusion.[[27]](#footnote-27)

Further investigation revealed that the supposed fusion site encodes a special region (DNA binding site) within a highly expressed gene.26 This makes sense of the earlier finding that this region aligned with many regions on different human chromosomes – since DNA binding sites are found in multiple locations around the human genome.  This gene had originally been characterized as a pseudogene, supposedly a broken gene.  Many pseudogenes are now known to be expressed and have been shown to carry out important regulatory functions.  The gene that the alleged fusion is located inside is highly expressed in many tissues throughout the human body. It is also highly networked with many other important genes expressed in the human body involved in blood cell development and cell signaling.  The alleged fusion site is really a functional feature in the human genome that helps regulate an important gene – it is not the result of a genomic accident, but has a special designed purpose.

**A More Plausible Explanation of the Evidence**

Based on this research, it appears that Dr. Miller’s snide suggestion that “that’s the way the designer made it”3 is actually a very sensible lay-level summary of the evidence.  The sequence does not match what would be expected by evolutionists, but instead encodes a portion of a highly expressed gene.  Obviously, this fits very nicely within a biblical worldview.  Sure, there could easily have been some changes to the sequence since the time of creation.  However, given its role today, it is functioning as something that was intelligently designed.

I recently visited Dr. Miller’s website,[[28]](#footnote-28) looking for an update by him on the alleged chromosome 2 fusion since I remembered how vigorously he promoted that concept. Alas, I didn’t find what I was looking for. Has he begun to doubt common ancestry between humans and apes?  It doesn’t look like it given that his website still promotes plenty of anti-creation and anti-ID material. This shows, as Ken Ham pointed out in his debate with Bill Nye, that it is not really about the evidence.  Everyone interprets evidence within a framework, or worldview.  If someone does not wish to consider the possibility of creation, there is no evidence that will ever convince them.

<https://answersingenesis.org/genetics/dna-similarities/chromosome-tales-and-importance-biblical-worldview/>

Gene Complexity Eludes a Simple Definition

by Jeffrey Tomkins, Ph.D. \*

In the early days of molecular genetics in the 1960s and ‘70s, it was widely held that a gene could be defined as a single entity that encodes the information to make a protein. However, as genetic studies have progressed, our understanding of what defines a gene has become incredibly more complicated.1 We still hear evolutionists claim “this and that creature have the same genes and are therefore related through common descent in evolution,” but in light of recent genetic studies, this claim is grossly oversimplified.

First, the boundaries of what can be called a single gene are becoming increasingly hard to define, along with its complete set of functions. Entire chromosomes and genomes are a continuum of pervasive and overlapping transcription (copying DNA into RNA).2,3Recent discoveries have revealed that the genes of many plants and animals are not like single entities at all but are rather a mixture of genes within genes and even genes that overlap each other.3 The regulatory control regions of genes, called promoters, can be shared by two completely different genes running in opposite directions from each other. (Genes are found on both strands of the double-stranded DNA molecule.) Enhancer regions that also play an important role in regulating gene function can be up to a million bases away from the gene they regulate. As if this weren’t enough, many genes function both forward and backward at the same time—producing both sense and antisense transcripts!4 The regulatory sequences of genes can also be located inside other nearby genes, and researchers have determined that genes dynamically interact with each other in “gene neighborhoods” much more than previously believed, to the point of blurring the boundaries between them.

Secondly, the informational output provided by genes can change depending on different circumstances. These circumstances include cell type, tissue type, and other stimuli such as the external environments.5 In the genome, both the DNA molecule itself and the histone proteins that the DNA molecule is packaged around can be chemically altered or tagged. The study of these chemical tags is called *epigenetics*or*chromatin remodeling*.5 In addition to genes having overlapping boundaries and alternate functions, the information provided by the genes is epigenetically altered by the cellular machinery to provide just the right output for the situational need at hand.

When evolutionists talk about creatures sharing the same genes, they are typically referring to very small segments of DNA in the genome. And in most cases, they are only referring to the small pieces of protein-encoding genes called exons—not the whole segment of DNA that is actually responsible for producing the information to make the correct version of the protein at the right time and in the correct amount.

But what about all the other expressed DNA sequences in the genome besides protein-coding segments—can they be called genes too? Amazingly, there are actually more than twice as many long non-coding RNA genes in the human genome as there are protein-coding genes, and these are turning out to be the key factors in what controls and regulates protein-coding genes, and in what also makes different kinds of creatures genetically unique or distinct from each other.6

Because of what we now know about the genome, you should be aware that when someone uses the term gene, the situation is a whole lot more complicated than it used to seem. To quote Dorothy from the classic movie *The Wizard of Oz*, “I’ve a feeling we’re not in Kansas anymore.” The biocomplexity of the genome is now reaching proportions beyond humankind’s wildest imaginations. An omnipotent Creator is the only possible explanation for such vast and elegant engineering.

***References***

1. Portin, P. 2009. The elusive concept of the gene. *Hereditas*. 146 (3): 112-117.
2. Djebali, S. et al. 2012. Landscape of transcription in human cells. *Nature*. 489 (7414): 101-108.
3. Clark, M. B. et al. 2013. The dark matter rises: the expanding world of regulatory RNAs. *Essays in Biochemistry.* 54: 1-16.
4. Antisense transcripts are believed to help regulate the genes from which they are copied in reverse—possibly by binding to the sense RNA transcripts directly or by facilitating the splicing and processing of the sense transcripts. See Pelechano, V. and L. M. Steinmetz. 2013. Gene regulation by antisense transcription. *Nature Reviews Genetics.*14 (12): 880-893.
5. Zhu, J. et al. 2013. Genome-wide chromatin state transitions associated with developmental and environmental cues. *Cell*. 152 (3): 642-654.
6. Managadze, D. et al. 2013. The vast, conserved mammalian lincRNome.*PLoS Computational Biology.*9 (2): e1002917.

*\* Dr. Tomkins is Research Associate at the Institute for Creation Research and received his Ph.D. in genetics from Clemson University.*

Cite this article: Jeffrey Tomkins, Ph.D. 2014. [Gene Complexity Eludes a Simple Definition](http://www.icr.org/article/8129/). *Acts & Facts*. 43 (6).

1. Pam Sheppard, “[Dover, Pennsylvania (USA) Intelligent Design trial ends today](https://answersingenesis.org/docs2005/1104dover.asp),” Answers in Genesis, http://www.answersingenesis.org/docs2005/1104dover.asp. [↑](#footnote-ref-1)
2. F. Morel et al., [Meiotic segregation of translocations during male gametogenesis](http://www.ncbi.nlm.nih.gov/sites/entrez?cmd=Retrieve&db=PubMed&list_uids=15271199&dopt=AbstractPlus), *Int. J. Androl.* 27 no. 4 (2004):200–212. [↑](#footnote-ref-2)
3. There are patterns of DNA that generally occur at the end of chromosomes which appear in the middle of chromosome 2 where the fusion is believe to have occurred (subtelomeric duplications). While there is no second centromere, there are patterns of DNA found near centromeres that occur in chromosome 2 where the second centromere is believed to have previously existed (pericentromeric duplications). [Hillier, L.W., et. al., Generation and annotation of the DNA sequences of human chromosomes 2 and 4, *Nature* 434 no. 7034 (2005):724–731.](http://www.bx.psu.edu/miller_lab/dist/chr2.4.pdf)   
   The possibility of human chromosome 2 being the result of a fusion is not a problem for creationists. It is only the idea that this chromosome was derived from a nonhuman ancestor that we would take issue with. [↑](#footnote-ref-3)
4. [Ken Miller on Human Evolution](http://www.youtube.com/watch?v=zi8FfMBYCkk), http://www.youtube.com/watch?v=zi8FfMBYCkk. [↑](#footnote-ref-4)
5. Gen 1:26–28; 2:7 [↑](#footnote-ref-5)
6. David Catchpoole, “[Petulant parrot proves a point—but atheists can’t (or won’t) see it](http://www.creationontheweb.com/content/view/4592/),” Creation Ministries International, http://www.creationontheweb.com/content/view/4592/; Ryan Jaroncyk, “[Parrot prodigy](http://www.creationontheweb.com/content/view/4901/),” Creation Ministries International, http://www.creationontheweb.com/content/view/4901/. [↑](#footnote-ref-6)
7. Ryan Jaroncyk, “[Jumbo Minds](http://www.creationontheweb.com/content/view/4713),” Creation Ministries International, http://www.creationontheweb.com/content/view/4713/; A. A. S. Weir, et al., “[Shaping of Hooks in New Caledonian Crows](http://www.sciencemag.org/feature/data/crow/),” AAAS, http://www.sciencemag.org/feature/data/crow/. [↑](#footnote-ref-7)
8. Chromosomal rearrangement may play some role in speciation, but it is not associated with the type of major anatomical rearrangements that ape to human evolution demands. They also come at a cost, since some DNA is generally lost during the rearrangements. Also, some rearrangements are associated with abnormalities. For example, it is estimated that 5% of Down’s syndrome cases are the result of an extra 21st chromosome carried on a translocation. Ref. 10. [↑](#footnote-ref-8)
9. M.W. Nachman and Myers, P., “[Exceptional chromosomal mutations in a rodent population are not strongly underdominant](http://www.pnas.org/cgi/reprint/86/17/6666),” *PNAS* 86 no. 17 (1989): 6666–6670. [↑](#footnote-ref-9)
10. J.K. Lightner, “[Changing chromosome numbers](http://www.creationontheweb.com/images/pdfs/tj/j20_3/j20_3_14-15.pdf),” *Journal of Creation* 20 no. 3 (2006):14–15. [↑](#footnote-ref-10)
11. A.N. Bruere and Ellis, P.M., “[Cytogenetics and reproduction of sheep with multiple centric fusions (Robertsonian translocations)](http://www.ncbi.nlm.nih.gov/sites/entrez?Db=pubmed&Cmd=ShowDetailView&TermToSearch=513026&ordinalpos=6&itool=EntrezSystem2.PEntrez.Pubmed.Pubmed_ResultsPanel.Pubmed_RVDocSum),” *J. Reprod. Fertil.* 57 no. 2 (1979):363–375. [↑](#footnote-ref-11)
12. E. Berezikov et al., “[Diversity of microRNAs in human and chimpanzee brain](http://www.ncbi.nlm.nih.gov/sites/entrez?cmd=Retrieve&db=PubMed&dopt=AbstractPlus&list_uids=17072315),” *Nature Genetics* 38 no. 12 (2006):1375–1377. [↑](#footnote-ref-12)
13. P. Borger and Truman, R., “Ultraconserved sequences pose megaproblems for evolutionary theory,” *Journal of Creation* 21 no. 2 (2007):8–9. [↑](#footnote-ref-13)
14. While it may turn out that this is not the case, it is fully consistent with a straightforward interpretation of the Bible. Nothing in Scripture implies that God must have created different kinds with different chromosome numbers or even different banding patterns. [↑](#footnote-ref-14)
15. “[A Victory for Science and Education in Dover](http://www.millerandlevine.com/dover/index.html),” http://www.millerandlevine.com/dover/index.html. [↑](#footnote-ref-15)
16. Roger Patterson, *Evolution Exposed,* (Petersburg, Kentucky: Answers in Genesis, 2006), [chapter 1](https://answersingenesis.org/articles/ee/what-is-science). [↑](#footnote-ref-16)
17. R. Bandyopadhyay et al., “[Parental Origin and Timing of De Novo Robertsonian Translocation Formation](http://www.journals.uchicago.edu/AJHG/journal/issues/v71n6/024289/024289.web.pdf),” *Am. J. Hum. Genet.* 71 no. 6 (2002):1456–1462. [↑](#footnote-ref-17)
18. Gametes normally contain one of each chromosome pair. When there is a loss or gain of chromosomal material during formation, an unbalanced gamete results. This can be from a missing chromosome, or from a translocated chromosome occurring along with one (or both) of its homologues. If a translocation is present without either homologue, then the gamete will be balanced. [↑](#footnote-ref-18)
19. Lightner, J. 2007. [A Tale of Two Chromosomes](https://answersingenesis.org/genetics/dna-similarities/a-tale-of-two-chromosomes/), *Answers in Depth* 2, answersingenesis.org/genetics/dna-similarities/a-tale-of-two-chromosomes/ [↑](#footnote-ref-19)
20. [www.youtube.com/watch?v=8FGYzZOZxMw](http://www.youtube.com/watch?v=8FGYzZOZxMw); [www.youtube.com/watch?v=zi8FfMBYCkk](http://www.youtube.com/watch?v=zi8FfMBYCkk) [↑](#footnote-ref-20)
21. www.youtube.com/watch?v=zi8FfMBYCkk [↑](#footnote-ref-21)
22. Bergman, J. and J. Tomkins. 2011. The chromosome 2 fusion model of human evolution – part 1: re-evaluating the evidence.  *Journal of Creation* 25, no. 2:106–110. [↑](#footnote-ref-22)
23. Ijdo, J. W., et al. 1991. Origin of human chromosome 2: an ancestral telomere-telomere fusion. *PNAS* 88:9051–9055. [↑](#footnote-ref-23)
24. Tsipouri, V. 2008. Comparative sequence analyses reveal sites of ancestral chromosomal fusions in the Indian muntjac genome.  *Genome Biology* 9, no. 10:R155. [↑](#footnote-ref-24)
25. The idea that the arrays are degenerate is an inference based on assuming the region resulted from a fusion of primate chromosomes; thus the authors carried in the belief that the arrays were once there, consistent with interpreting the data within an evolutionary framework.  Fan, Y. et al. 2002. Genomic structure and evolution of the ancestral fusion site in 2q13-2q14.1 and paralogous regions on other human chromosomes.  *Genome Research*12, no. 11:1651–1662. [↑](#footnote-ref-25)
26. Tomkins, J. P. 2013. Alleged Human Chromosome 2 “Fusion Site” encodes an active DNA binding domain inside a complex and highly expressed gene – negating fusion.  *Answers Research Journal* 6:367–375. [↑](#footnote-ref-26)
27. Tomkins, J and J. Bergman. 2011. The chromosome 2 fusion model of human evolution – part 2: re-analysis of the genomic data.  *Journal of Creation* 25, no. 2:111–117. [↑](#footnote-ref-27)
28. [www.millerandlevine.com/km/evol](http://www.millerandlevine.com/km/evol/) [↑](#footnote-ref-28)