

An Evangelical Geneticist's Critique of *Reasons to Believe's* Testable Creation Model: RTB and Human-Ape Common Ancestry

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*"One serious critique of young-earth creationist attempts to explain the natural realm is that their explanations, typically rooted in religious dogma, have no flexibility to adapt and self-correct as knowledge increases."*¹

Hugh Ross

Introduction

Reasons to Believe (RTB) is the most influential Old-Earth Creationist organization in North America. While RTB supports a mainstream scientific position on cosmology and the age of the earth, RTB rejects evolutionary biology. Specifically, RTB denies that humans share ancestry with other forms of life, such as Neandertals or chimpanzees. RTB also claims that all human are the descendents of a single, specially created couple who lived about 50,000 years ago. RTB has expounded a framework called the "Testable Creation Model" in three major books published in the last five years: *Who Was Adam?* was published in 2005; *Creation as Science* was published in 2006, and *More Than a Theory* was published in 2009.² Furthermore, RTB claims that this model is scientifically robust. This same period however, has also seen the publication of much genetic data relevant to assessing human common ancestry.³ This paper will examine the interaction between RTB literature and several lines of genetics-based evidence for common ancestry. In so doing, I will address the scientific robustness and reliability of the RTB model. RTB welcomes such critique from qualified scholars in their works as a means of improving their model.⁴ This critique, while forthright, is offered without animosity and in good faith. It is my hope that RTB will find it useful for correcting several serious flaws in their approach to human origins.

A recent history of primate comparative genomics

When the human genome project (the endeavor to determine the complete DNA sequence of every human chromosome) was completed in 2003, the equivalent chimpanzee genome project was already underway. Just prior to the completion of the human project (from about 2002 on), detailed comparisons of large stretches of DNA between humans and chimpanzees became possible as both genome projects progressed. As the data came in, a range of estimates for the precise amount of identity between the two genomes was published in the mainstream scientific literature (Table 1 on next page).

Such estimates took two forms: measuring single-nucleotide differences in sequences found in both genomes while omitting inserted or deleted sequences (collectively called "indel" mutations, because it may be difficult to determine if a difference is due to an insertion or deletion), or estimates combining both sources of variation. In the years preceding the completion of the chimpanzee genome in 2005, partial-genome comparisons repeatedly estimated the two genomes to be over 98% identical when omitting differences due to indels. Two pre-2005 studies took indels into consideration as well: Britten (2002) estimated the two genomes to be 95% identical, whereas Anzai *et al.* (2003) found only 87% identity. This paper examined a chromosomal region that contains immune system genes, and was not thought to be representative of the genome as a whole. This prediction was borne out in 2005 when the completed human and chimpanzee genomes were compared (2.9×10^9 DNA base pairs). The final tally was 98.77% identical when indels were included and 95% identical when indels were omitted. A second paper published in 2005 examined 1.85×10^7 DNA base pairs found in the portions of the chromosome that specify proteins, and found even higher identity (99.4%) in these sequences.

Date	Author	Sample size (DNA base pairs)	Homology (excluding indels)	Homology (including indels)	WWA (2005)	CAS (2006)	MTT (2009)	Discussed / cited in RTB books?	Notes & Comments
1996	Arnason et al.	1.65 x 10 ⁵	91.10%	91.10%	Yes	Yes	Yes		1st complete <i>mitochondrial</i> DNA comparison; conflated with genomic studies in CAS and MTT
2002	Ebersberger et al.	1.9 x 10 ⁶	98.76%	NC	Yes	No	No		Largest study available at time of publication; not mentioned in in CAS or MTT
2002	Britten	7.79 x 10 ⁵	98.6%	95%	Yes	No	No		1st large study to examine homology with/without indels, not mentioned in in CAS or MTT
2003	Anzai et al.	1.87 x 10 ⁶	98.6%	87%	Yes	Yes	Yes		Paper examining the MHC cluster (a region with unusually numerous indels); claimed as best estimate of the entire genome in RTB literature
2003	Thomas et al.	1.8 x 10 ⁶	98.85%	NC	Yes	Yes	Yes		Paper examining homology for one genomic region across multiple vertebrate species
2004	Consortium paper	3.33 x 10 ⁶	98.56%	NC	Yes	Yes	Yes		Results of sequencing and comparing chimpanzee chromosome 22 with the human genome; largest study to date at time of publication
2005	Consortium paper	2.7 x 10 ⁹	98.77%	95%	NA	No	No		Landmark paper comparing the completed chimpanzee genome with the human genome; largest study to date (sample size is three orders of magnitude greater than previous work); not mentioned in RTB literature
2005	Nielson et al.	1.85 x 10 ⁷	99.4%	NA	NA	No	No		Genome-wide, human / chimpanzee comparison restricted to protein-coding sequences; not mentioned in RTB literature

NC = not calculated
NA = not applicable

WWA = *Who Was Adam?* (2005)
CAS = *Creation as Science* (2006)
MTT = *More Than a Theory* (2009)

The RTB model and comparative primate homology: overview

A key tenet of the RTB model is that humans do not share ancestry with other forms of life. As such, RTB has invested considerable effort in reinterpreting human / chimpanzee genomic homology comparisons for their constituents. A notable feature of the RTB model is the claim that the whole-genome, human / chimpanzee homology value is in fact 85-90%, not 95-99%.⁵ As we have seen, comparisons between the human and chimpanzee genomes progressively improved in the early 2000s, culminating in the landmark whole-genome comparison of 2005. The fact that this data emerged over time allows us to investigate how RTB responded over the same time period, and as such examine the reliability of the RTB model as new data became available that were at odds with one of its non-negotiable claims.

The RTB model and primate genomics (2005): *Who Was Adam?*

Like comparative primate genomics, the RTB creation model also hit a milestone in 2005 with the publication of *Who Was Adam?* (WWA) by RTB scholars Fazale Rana and Hugh Ross.⁶ This book narrowly predates the pivotal 2005 whole-genome comparison paper. Unlike two later RTB books (see below) this

book discusses research-to-date on human-chimpanzee comparative genetics in extensive detail.⁷ *WWA* carefully distinguishes between estimates based on including or excluding indels, as well as chromosomal or mitochondrial DNA. However, *WWA* made a questionable claim when it states:

*The most comprehensive genetic comparisons indicate that humans and chimpanzees share genetic similarity closer to about 85 percent than to 99 percent. From an evolutionary perspective, if a 99 percent genetic similarity reflects a close evolutionary connection, then an 85% genetic similarity distances humans from chimpanzees.*⁸

This assertion, however, was already at odds with the conclusions of the 2004 Chromosome 22 Consortium paper (Table 1), a paper that is cited in *WWA* as support for differences in human-chimpanzee gene expression.⁹ At the time *WWA* was published, RTB expected future genomic comparisons to widen the gap between humans and chimpanzees.¹⁰

As we will soon see, they did not have long to wait. Their hope of establishing greater dissimilarity, however, was in vain.

The RTB model and primate genomics (2006): *Creation as Science*

In early September 2005, a full comparison between the completed chimpanzee genome and the human genome was published in the prestigious journal *Nature*. This landmark paper used a sample size of 2.9×10^9 base pairs: it covered virtually the entire genomes of both species, dwarfing previous comparisons (Table 1). This comparison returned results consistent with previous studies: homology excluding indels was over 98%; including indels brought it down to 95%. As expected, the 2003 Anzai et al., paper (that found 87% homology including indels in one chromosomal region) was shown to be an inappropriate estimate for the genome as a whole. Beyond its wide impact in the biological sciences, this paper also received much attention from the mainstream media. This work, however, did not make any discernable difference to the RTB model. In 2006, another major RTB book, *Creation as Science (CAS)*, appeared. In contrast to the lengthy, detailed discussion of human / chimpanzee comparative genomics in *WWA*, *CAS* has only a brief section as follows:

*New research, however, indicates that the widely advertised 98 to 99 percent similarity between human and chimpanzee DNA is greatly exaggerated. Such claims are based on small segments of the human and chimpanzee genomes where common sense dictates that the similarities would be the greatest. While comparisons between the complete human genome and the complete chimpanzee genome have only recently begun, the most complete comparisons performed thus far indicate that the degree of similarity is more like 85 to 90 percent.*¹¹

The above paragraph from *CAS* cites four research publications, each of which was previously cited in *WWA*: Anzai et al., 2003; Thomas et al, 2003; Arnason et al., 1996; and the Chromosome 22 Consortium paper of 2004 (Table 1). Surprisingly, *CAS* makes no mention of the actual whole-genome study published between *WWA* and *CAS*. On encountering this, I initially assumed that Ross and Rana were simply unaware at the time *CAS* went to press that the chimpanzee genome had been completed, or that perhaps they had mistaken the Anzai paper as a whole-genome analysis. Further investigation, however, failed to support

these hypotheses. First, in an article published in 2004 in the RTB periodical *Connections*, Rana emphasizes that the Anzai paper is not a whole genome comparison and discusses its actual data set in detail:

Though these whole-genome comparisons are not yet possible, scientists are close, and preliminary results indicate that humans and chimpanzees are really not so genetically similar... another study found only 86.7% genetic similarity when segments of human and chimpanzee DNA (totaling 1,870,955 base pairs) were laid side by side.¹²

Thus Rana correctly understands that the Anzai paper is not a whole-genome comparison. Secondly (and more significantly), Rana does mention the key 2005 whole-genome paper in the very first 2006 edition of *Connections*, and notes correctly that it is a whole-genome study:

Where were you on September 1, 2005? Perhaps you missed the announcement of a scientific breakthrough: the influential journal Nature published the completed sequence of the chimpanzee genome. This remarkable achievement received abundant publicity because it paved the way for biologists to conduct detailed genetic comparisons between humans and chimpanzees.¹³

Rana uses this as an introduction to discuss another article from the same journal issue and does not discuss this key paper or its results, beyond a footnote referring readers to *WWA*. While this *Connections* article does not have a precise publication date, Rana cites accession of online material for this article as occurring on November 30, 2005. Other articles in this edition of *Connections* also cite access dates in late November 2005, suggesting that this volume was drafted in late 2005 for publication early in 2006. The chapter in *CAS* discussing human / chimpanzee genomics has references to online material cited as accessed in April 2006, indicating that this chapter was still in revision at this time. Taken together, these lines of evidence strongly suggest that RTB was aware of the key 2005 paper at the time the *CAS* chapter was in preparation, and that they correctly understood its significance as the first whole-genome comparison. The choice of language in *CAS* also supports this conclusion, since the chapter claims that “... comparisons between the complete human genome and the complete chimpanzee genome have only recently begun...” which makes sense only if both genomes were sequenced at the time of its writing. Despite this concession, *CAS* makes no mention of the key 2005 paper or its findings, and claims rather that “the most complete comparisons performed thus far” support homology values in the 85-90% range. In reality, only the Anzai paper, which covered a small chromosomal region expected to be disproportionately different between the two species, supports this value (Table 1).

The RTB model and primate genomics (2009): *More Than a Theory*

The next major RTB publication dealing with human / chimpanzee genomic comparisons was *More Than a Theory (MTT)* published in 2009. The relevant passage in *MTT* is a lightly reworked version of what appears in *CAS*, with only one notable change: whereas *CAS* acknowledges that comparisons between the completed genomes are underway, *MTT* claims they have not yet been done (Figure 1).

Like *CAS*, *MTT* claims “the most complete analyses performed so far” indicate homology values in the 85-90% range, makes no mention of the key 2005 whole-genome comparison paper, and again cites exactly the same references as *CAS*, the most recent being the 2004 consortium paper (Table 1). Thus, four

years after they were aware of the key 2005 paper, there is still no mention of it to be found in the RTB framework; moreover, *MTT* claims such an analysis has never been performed.

“New research, however, indicates that the widely advertised 98 to 99 percent similarity between chimpanzee and human DNA is greatly exaggerated. Such claims are based on small segments of the human and chimpanzee genomes where common sense dictates that the similarities would be the greatest. While comparisons between the complete human genome and the complete chimpanzee genome **have only recently begun**, the most complete comparisons performed thus far indicate that the degree of similarity is more like 85 to 90 percent.”

Creation as Science (2006) p. 156

“New research, however, indicates that the widely advertised 98 to 99 percent similarity between chimpanzee and human DNA is greatly exaggerated. Such claims were based on small segments of the human and chimpanzee genomes where common sense dictates that similarities would be the greatest. While comparisons between the complete human and chimpanzee genomes **have yet to be done**, the most complete analyses performed so far show that the similarity is closer to 85 to 90 percent.”

More Than a Theory (2009) pp. 187-188

The RTB model and primate genomics – present day

The pattern we have seen in the major RTB books continues into the present. Rana, for example, continues to argue that the best estimate of whole-genome human chimpanzee homology is at best 90%. For example, during a recent podcast discussion of the completed western clawed frog genome (*Xenopus tropicalis*)¹⁴ Rana claims: “*It’s common parlance that humans and chimps have a 99% genetic similarity. The actual data indicates probably it’s closer to 90% similarity as opposed to 99% similarity*” before going on to imply that the higher value can only be supported through comparisons of specific genes. He then goes on to claim that sequenced frog genome shows 80% similarity to the human genome based on “*the same reasoning.*” The argument he makes is an attempt to cast doubt on the relevance of the human / chimpanzee comparison: if humans and chimps are 90% similar and humans and frogs are 80% similar, Rana claims these “*are not meaningful comparisons in a biological sense.*” Rana’s argument, however, is deeply flawed in that he is comparing two very different measures of similarity and claiming they are equivalent. The human / chimpanzee value, as we have seen, is 95% genome-wide identity (including indels) for the completed genomes of both species compared across approximately three billion DNA base pairs (Table 1). The 80% value Rana touts for the human / frog comparison, however, *is merely a measure of the percentage of genes in the frog genome that have a similar gene in humans implicated in a human disease.*¹⁵ It is not even a measure of the genetic similarity of those genes, but merely a fraction of the genes identified in frog that might be useful for studying human diseases. Rana, however, presents these two values as equivalent measures in an attempt to downplay human / chimpanzee genomic similarity. In reality, the genome-wide homology between *Xenopus tropicalis* and humans is slightly over 30%.¹⁶

Taken together, these findings demonstrate the following: (a) RTB carefully followed the primary literature on human / chimpanzee comparative genomics up until and including a major paper published in 2004, even if it represented such studies selectively to their constituents; (b) RTB was aware of the key 2005 whole-genome study and correctly understood its implications at the time the first 2006 edition of

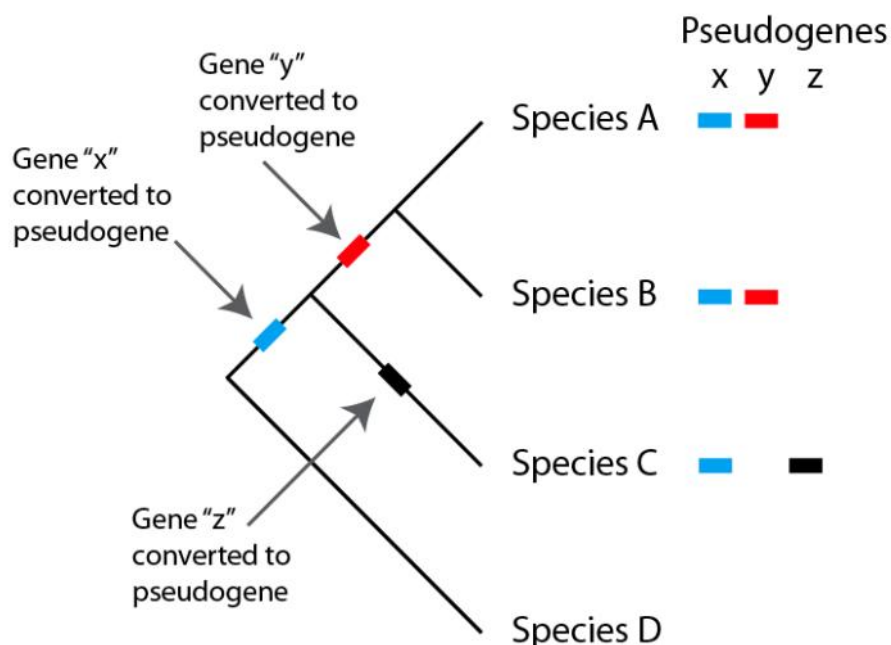
Connections was drafted in late 2005; (c) RTB has made no mention of this paper (nor any paper in this field published since 2004) in two major works published after this paper was available; (d) RTB continues to claim, five years after this paper was published, that the most recent and most extensive evidence supports their preferred value of 85-90% homology (and that higher values can only be supported with small, biased samples), despite the fact that this conclusion is starkly at odds with the best and most extensive study available, and is itself derived from a comparatively small, biased sample; and (e) RTB has shifted from acknowledging (in 2006) that whole-genome comparisons have been done to denying (in 2009) that they ever have.

The RTB Model and Pseudogenes

Pseudogenes (literally, “false genes”) are the remnants of once-functional genes that persist in genomes after they lose function. Pseudogenes are often shared among species in a nested pattern that strongly supports common ancestry.¹⁷ Additionally, the human genome harbors pseudogene remnants of genes devoted to non-mammalian ways of life.¹⁸ For those unfamiliar with this line of evidence for evolution, Darrel Falk and I have recently written about pseudogenes in the human genome for a lay audience.¹⁹ Since shared pseudogenes are such clear indicators of common ancestry, RTB has also expended significant effort on discussing pseudogene data in their major works. As such, pseudogene evidence is a second opportunity to test the scientific reliability of the RTB model.

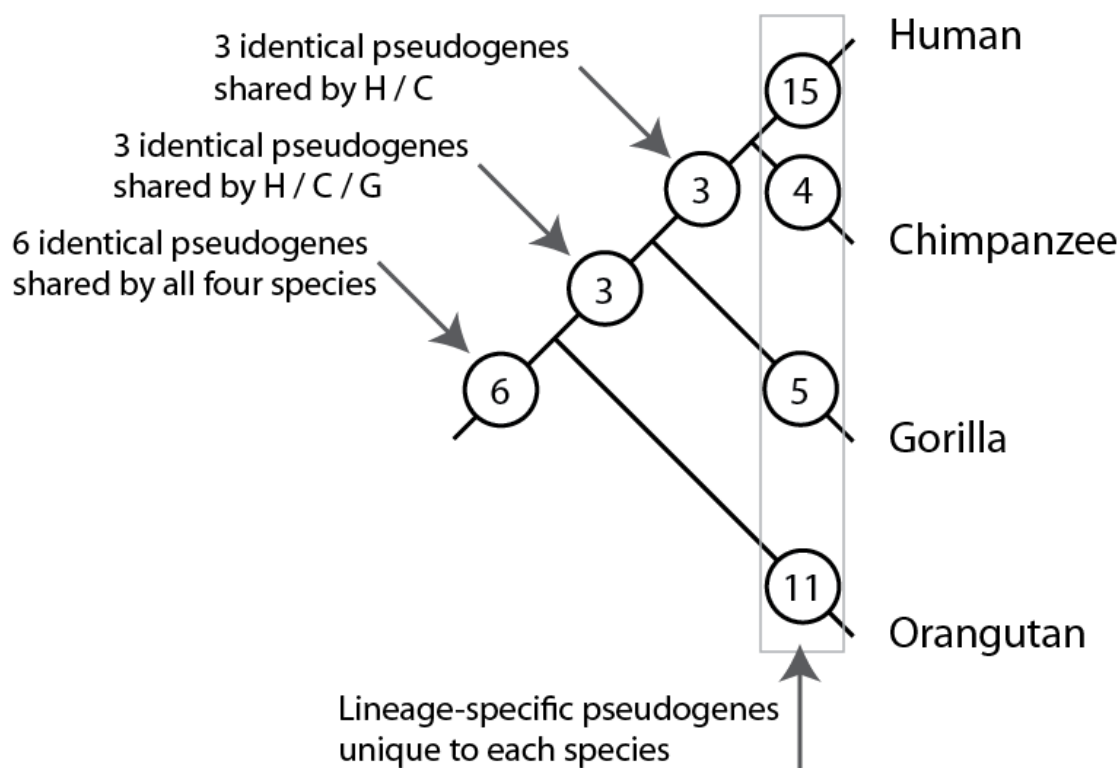
Understanding the difference between two specific classes of pseudogenes is important for evaluating the RTB literature: specifically, *processed* versus *unitary* pseudogenes. Processed pseudogenes are the remains of RNA copies of genes that have inserted themselves into chromosomes, whereas unitary pseudogenes are the remains of genes that have been inactivated due to mutation. In the case of processed pseudogenes, the original gene remains intact and functional.

One of the most compelling features of pseudogene evidence for common ancestry is that pseudogenes (of all kinds) form *nested hierarchies*. Consider an example phylogeny (evolutionary history, or family tree) for four modern species (A, B, C and D) given in Figure 2.



Phylogenies can be assembled using genome sequence similarity (grouping more closely related organisms together) as well as through other measures, such as anatomy. A phylogeny assembled through these criteria (which agree and reinforce one another) can be used to predict what the distribution of various pseudogenes will be in the four modern species. For example, pseudogenes from the very distant past (and thus present in the common ancestor of the whole group) will appear in all modern species, with the same mutations. Pseudogenes shared by species A and C (e.g. the pseudogene form of “gene X” in the diagram) are best explained as having been present in their common ancestor. Since the common ancestor of A and C is also the common ancestor of species B, observing a shared pseudogene between A and C makes a precise prediction: if the species do share common ancestry and we have the phylogeny correct, species B should have the same pseudogene with the same mutation. The pseudogenes should be present in a nested hierarchy. Note too that mutations may happen in a lineage after its last known speciation event (e.g. “Gene Z” in species C). In this case, this pseudogene should be unique to that species: we should not find it in the other species in the phylogeny.

One example of a nested, hierarchical pattern of unitary pseudogenes in primates is that of olfactory receptor pseudogenes.²⁰ An analysis of these pseudogenes in humans, chimpanzees, gorillas and orangutans produces a nested hierarchy that independently groups these species into a phylogeny identical to the one assembled from sequence homology data: humans share the most pseudogenes in common with chimpanzees, less pseudogenes in common with gorillas, and so on (Figure 3).



Most importantly, the nested hierarchical pattern is not violated: for example, all the pseudogenes present in both gorillas and humans (and thus in the common ancestor of these species) are present in

chimpanzees (since the common ancestor of humans and gorillas is also the common ancestor of chimpanzees). Not one of these pseudogenes is out of place. A second, equally powerful line of pseudogene evidence for common ancestry is the presence of pseudogenes that show adaptation to manners of life that do not make sense for the organism in question. Whereas humans do need (at least some) olfactory receptors, the human genome contains pseudogene remnants of genes that mammals do not need. One example that I have discussed previously is the *vitellogenin* pseudogene found in the human genome.²¹ Vitellogenin is a protein component of egg yolk, and as such is a functional gene in amniotic (egg-laying) organisms. Humans are of course *placental* mammals, yet we have the *vitellogenin* gene present in our genomes as a unitary pseudogene. The two functional genes flanking the human *vitellogenin* pseudogene are the same two genes flanking the functional *vitellogenin* gene in chickens. These data make perfect sense if humans are descended from egg-laying ancestors and share common ancestry with chickens. It is very difficult to rationalize this data from an antievolutionary perspective. Since the common ancestor of humans and chickens was a reptile, this indicates that the *vitellogenin* pseudogene should be present in all non-egg-laying mammals. Studies so far have found this unitary pseudogene in wide variety of additional species ranging from dogs to wallabies. As expected, egg-laying mammals such as the platypus retain a functional version of this gene.²²

Unitary pseudogenes present in nested hierarchies that independently group organisms into phylogenies assembled with other data are incredibly powerful evidence for common ancestry. Additionally, the fact that the genomes of multiple placental mammals (including humans) contain a unitary pseudogene clearly adapted for egg laying (in the precise genomic location predicted by common ancestry), is very challenging to explain from an antievolutionary perspective. Accordingly, any attempt to scientifically refute common ancestry must address these types of evidence in a convincing manner.

The most extensive discussion of pseudogene evidence for common ancestry in the RTB literature is found in *WWA*, where an entire chapter is devoted to the topic.²³ While *WWA* does not specifically address the fact that pseudogenes are observed in nested hierarchies, it does at least mention that unitary pseudogenes with identical mutations are shared between primates, including humans and chimpanzees.²⁴ The more recent RTB books, however, make no mention of these data when discussing pseudogenes. The core of RTB's attempt to refute pseudogene evidence in all three books is their claim that pseudogenes are functional sequences: "*Non-coding DNA regions (including pseudogenes, LINES, SINEs and endogenous retroviruses) aren't really junk after all. These elements possess function.*"²⁵

The evidence offered for this assertion are examples of pseudogenes and other "junk DNA" that have been shown to have function, specifically processed pseudogenes, SINEs and LINES that have been implicated in gene regulation.²⁶ This argument, however, is scientifically weak: rare examples of processed pseudogenes and repetitive DNA elements such as LINES and SINEs that have retained or gained a function does not confer similar functionality on the many thousands of such sequences for which there is good evidence that they are not functional. Beyond this weakness is a more serious flaw: evidence for *unitary* pseudogene function is lacking. In *WWA*, RTB candidly admits that evidence for unitary pseudogene function is not to be found:

*"What about the genetic material without a known function, such as the GLO unitary pseudogenes that humans and chimpanzees share? Currently the RTB model offers no explanation for this feature. The model does predict, however, that as with other classes of noncoding DNA, function will one day be discovered for these uniting pseudogenes."*²⁷

The later RTB books, however, do not distinguish between unitary pseudogenes and other pseudogene / repetitive DNA classes. The same examples of rare functional processed pseudogenes, LINEs, SINEs and endogenous retroviruses are given, but unitary pseudogenes are not mentioned at all in either *CAS* or *MTT*.²⁸ Instead, the selected functional examples of non-unitary pseudogenes are used to imply that pseudogenes *in general* have been demonstrated to have function.²⁹ This is misleading. Unitary pseudogenes remain highly problematic for RTB, and becoming vague on this point does not overcome the difficulty they pose for the RTB model.

In summary, discussions of pseudogene evidence in the RTB model are selective, misleading and simply ignore the strongest lines of evidence that pseudogenes provide for common ancestry. The RTB approach to pseudogenes also suggests that the differences between *WWA* and the two more recent books may be a pattern: RTB is able and willing to provide extensive detail when the data are inconclusive, but becomes vague and imprecise when data that challenge the model become available.

Conclusions

As we have seen, the RTB model of human origins with respect to common ancestry is seriously flawed. It misrepresents well-established science, fails to address the strongest relevant evidence against its position, and selectively presents data in an attempt to support a pre-determined position that humans and other apes do not share ancestry. As such this model is not a model that a believer can hold with scientific integrity. It may well be that RTB offers their model in good faith: if so, however, it demonstrates that they are not qualified to address these lines of evidence in a scientific manner.

While this paper presents a careful analysis designed to substantiate my conclusions for a non-specialist audience, it should be noted that the scientific flaws in the RTB model are blatantly obvious to working scientists within the biological sciences. Moreover, scientists are likely to interpret these flaws as obfuscation or deliberate deception. As such, the packaging of such arguments with an overt Gospel message seriously compromises a Christian witness within this group and raises unnecessary barriers to faith. Within the community of believers, the extent to which one's faith is supported by such arguments is also the extent to which one is rendered vulnerable to crisis should those arguments fail: "today's reason to believe" sets one up for tomorrow's "reason to disbelieve" should the evidence be examined.

Notes

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2. Rana, Fazale and Ross, Hugh. *Who Was Adam? A Creation Model Approach to the Origin of Man*. Colorado Springs: NavPress, 2005; Ross, Hugh. *Creation as Science: A Testable Model Approach to End the Creation / Evolution Wars*. Colorado Springs: NavPress, 2006; and Ross, Hugh. *More Than a Theory: Revealing a Testable Model for Creation*. Grand Rapids: Baker Books, 2009.
3. For a review of these lines of evidence, see Venema, D.R. (2010). Genesis and the genome: genomics evidence for human – ape common ancestry and ancestral hominid population sizes. *Perspectives on Science and Christian Faith* 62 (3), 166-178.
4. *More Than a Theory*, p. 21.
5. *Who Was Adam?*, p. 223; *Creation as Science*, p. 156; *More Than a Theory*, p. 187-188.
6. Rana, Fazale and Ross, Hugh. *Who Was Adam? A Creation Model Approach to the Origin of Man*. Colorado Springs: NavPress, 2005.
7. *Ibid.*, pp. 212-215.
8. *Ibid.*, p. 223.
9. *Ibid.*, p. 222.
10. *Ibid.*, p. 223.
11. Ross, Hugh. *Creation as Science: A Testable Model Approach to End the Creation / Evolution Wars*. Colorado Springs: NavPress, 2006, p.156.

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13. Rana, Fazale. "First Chimpanzee Fossils Cause Problems for Evolution." *RTB Connections*, Vol. 8 (1), 2006. Emphasis in original. Available online at http://www.reasons.org/resources/publications/connections/2006q1#firs_chimpanzee_fossils_cause_problems_for_evolution
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17. Y. Gilad, O. Man, S. Paabo, and D. Lancet, "Human specific loss of olfactory receptor genes," *Proceedings of the National Academy of Sciences of the USA* 100 (2003): 3324-3327-5.
18. D. Brawand, W. Wali, and H. Kaessmann, "Loss of Egg Yolk Genes in Mammals and the Origin of Lactation and Placentation." *PLoS Biology* 6 (2006): 0507-0517.
19. Venema, D.R. and Falk, D. *Signature in the Pseudogenes* Parts 1 and 2. Available online at: <http://biologos.org/blog/signature-in-the-pseudogenes-part-2/> and <http://biologos.org/blog/signature-in-the-pseudogenes-part-1/>
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21. Venema, D.R. (2010). Genesis and the genome: genomics evidence for human – ape common ancestry and ancestral hominid population sizes. *Perspectives on Science and Christian Faith* 62 (3), 166-178.
22. D. Brawand, W. Wali, and H. Kaessmann, "Loss of Egg Yolk Genes in Mammals and the Origin of Lactation and Placentation." *PLoS Biology* 6 (2006): 0507-0517.
23. *Who Was Adam?*, pp. 226 – 243. The discussions in *Creation as Science* and *More Than a Theory* are brief summaries and introduce no new arguments.
24. *Who Was Adam?* pp. 228-230, 243
25. *Ibid.*, p. 235.
26. *Ibid.*, pp. 235-243.
27. *Ibid.*, p. 243.
28. *Creation as Science*, pp. 221-222; *More Than a Theory* p. 202.
29. *Ibid.*