

The book cover features a dark, starry background. In the foreground, a large, orange and red ringed planet, similar to Saturn, is shown. Behind the planet, there are faint, white geometric diagrams and lines, including circles, arcs, and a grid. A bright, glowing red star is visible in the upper left quadrant. The text is centered and white, with the title 'Intelligent Design 101' enclosed in a white rectangular box.

Leading Experts Explain  
the Key Issues

# Intelligent Design 101

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**H. WAYNE HOUSE**  
GENERAL EDITOR





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Appendix

A Reply to Francis Collins's  
Darwinian Arguments  
for Common Ancestry of  
Apes and Humans

CASEY LUSKIN AND LOGAN PAUL GAGE

**Note from the general editor:** This appendix is in response to the arguments for Darwinism and human-chimpanzee common ancestry as put forth by the scientist and Christian evolutionist Francis S. Collins in his widely discussed book, *The Language of God: A Scientist Presents Evidence for Belief* (New York: Free Press, 2006).

## Introduction

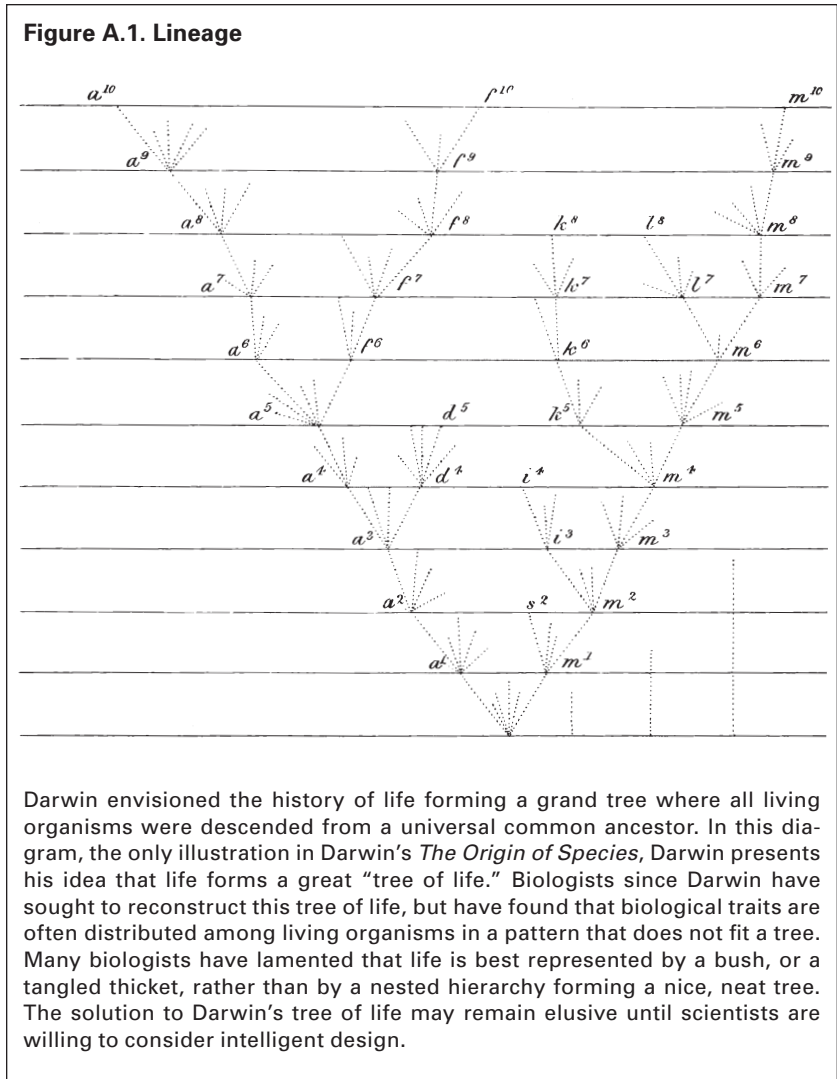
Francis Collins, respected scientist and director of the National Human Genome Research Institute, recently became a prominent figure in the evolution debate. As a self-described proponent of “theistic evolution”<sup>1</sup> and a Christian, Collins’s cachet is his ability to communicate his sympathetic viewpoint on religion.<sup>2</sup> Given the antireligious arguments of leading Darwinists such as Richard Dawkins, Collins’s voice is a valuable addition to the evolution debate. Unfortunately, his widely discussed 2006 book, *The Language of God: A Scientist Presents Evidence for Belief*,<sup>3</sup> advances several questionable arguments for a Darwinian account of human-ape common ancestry.

Many assume that if common ancestry is true, then the only viable scientific position is Darwinian evolution—in which all organisms are descended from a common ancestor via random mutations and blind selection. Such an assumption is incorrect: Intelligent design *is not necessarily incompatible with common ancestry*.<sup>4</sup> Even if all organisms on earth share a common ancestor, it does not follow that the primary mechanisms causing the differences between the species must be blind, unguided processes such as natural selection. Nonetheless, Darwin’s tree of life (see fig. A.1) is an “icon of evolution” and therefore deserves careful examination.<sup>5</sup>

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The authors thank Tim Standish for his helpful comments, which aided the crafting of this response.

Darwin's tree of life—the notion that all living organisms share a universal common ancestor—has faced difficulties in the past few decades. Some leading biologists believe that universal common descent cannot explain the beginning of the tree of life. As W. Ford Doolittle explains, “Molecular phylogenists will have failed to find the ‘true tree,’ not because their methods are inadequate or because they have chosen the wrong genes, but because the history of life cannot properly be represented as



a tree.”<sup>6</sup> Doolittle, a Darwinist, elsewhere writes that “there would never have been a single cell that could be called the last universal common ancestor.”<sup>7</sup> Doolittle attributes his observations to gene-swapping among microorganisms at the base of the tree. But as discussed in chapter 3, Carl Woese finds that such problems exist beyond the base of the tree: “Phylogenetic incongruities [conflicts] can be seen everywhere in the universal tree, from its root to the major branchings within and among the various taxa to the makeup of the primary groupings themselves.”<sup>8</sup> A recent study looking at animal relationships concluded, “Despite the amount of data and breadth of taxa analyzed, relationships among most metazoan phyla remained unresolved.”<sup>9</sup> Indeed, the Cambrian explosion, during which nearly all of the major living animal phyla (or basic body plans) appeared over 500 million years ago in a geological instant, raises a serious challenge to Darwinian explanations of common descent.<sup>10</sup>

Collins argues for common ancestry at the level of humans and apes rather than focusing on the lower branches of the tree of life. But does common ancestry hold up even at these higher branches? To be sure, if humans and apes share a common ancestor, that would not refute intelligent design. But Collins claims that the evidence for common human-ape ancestry via purely Darwinian means is particularly strong. It is this claim that will be scrutinized here.

To his credit, Collins avoids the usual simplistic argument of claiming that mere genetic similarity is solid evidence for common ancestry. Collins sees through this, noting that genetic similarity “alone does not, of course, prove a common ancestor” because a designer could have “used successful design principles over and over again.”<sup>11</sup> Collins is right.

To show how this argument fails by analogy, if one discovers two similar Buicks in a junkyard, one would not conclude one car descended from the other. Rather, one would conclude that intelligent engineers modified plans from the first Buick to make the second. In the same way, genetic similarity between apes (particularly, chimpanzees) and humans—in itself—is compatible with either common descent or common design. For this reason, Collins offers four supplementary arguments for human-ape common ancestry and a Darwinian explanation of human origins:

1. human chromosomal fusion;
2. “junk” DNA in humans and apes;

3. non-coding DNA sequences that have more differences than protein-coding sequences of DNA; and
4. mutations that allegedly caused the evolution of human cognition and language.

## Human Chromosomal Fusion

One of the most frequent arguments in favor of human-ape common ancestry is that only common ancestry explains the fact that human chromosome 2 closely resembles chimpanzee chromosomes 2a and 2b.<sup>12</sup> Humans have twenty-three pairs of chromosomes, or forty-six total. Apes such as the chimpanzee have twenty-four pairs of chromosomes, or forty-eight total. Darwinists contend that humans have one less pair of chromosomes than apes because somewhere in the human line, two ape chromosomes became fused into one chromosome. Human chromosome 2 has a structure similar to what one would expect if two chromosomes resembling chimpanzee chromosomes 2a and 2b were fused to one another, end to end. Collins and many other Darwinists claim that this evidence demonstrates common ancestry.

Anyone who compares chimps and humans recognizes a high level of similarity in body structure (also called morphology). Indeed, pre-Darwinian scientists such as Carolus Linnaeus (1707–1778) recognized similarities between species and classified organisms accordingly without assuming common ancestry. Since similar body structures are usually built using similar genes, it is not surprising to learn that our genes are very similar to those of chimps.

We accept that there is good evidence that human chromosome 2 is composed of two fused chromosomes. It seems clear that a chromosomal fusion event took place at some time in our human lineage. This evidence, however, merely confirms something we already knew: humans and chimps have a similar genetic structure. As Collins reminds us, genetic similarity does not prove common ancestry, for genetic similarity may be the result of functional requirements, in this case possibly implicating common design.<sup>13</sup>

Evidence for chromosomal fusion in humans simply indicates that, at some point within our human lineage, two chromosomes became fused. This tells us nothing about whether we share a common ancestor with apes.



Neither Collins nor any other Darwinist of whom we are aware speculates that this fusion event caused our ancestors to *become* human. The fusion evidence merely tells us that our ancestors—whether essentially human or otherwise—once had forty-eight chromosomes. It does not tell us whether our ancestors were related to modern apes.

### **Predictions of Comparative Morphology?**

The Darwinist might respond, “But this evidence shows that our ancestors once had forty-eight chromosomes, just like chimpanzees and other apes. We would expect to see a fused chromosome in humans because that explains the data with the least number of genetic changes in species over evolutionary time. Moreover, our fused chromosome 2 contains segments resembling ape chromosomes 2a and 2b. Evolution would have predicted this evidence, so this confirms that prediction and demonstrates common descent.” While they may be correct that a prediction of Darwinian theory has been fulfilled, our chromosomal fusion could have been expected based upon observations and arguments that have nothing to do with Darwinism.

The Darwinist response merely restates the fact that humans and apes (like the chimpanzee) share a similar genetic structure. But long before the evidence for human chromosomal fusion was discovered, we knew about the high genetic similarity between humans and chimps. Moreover, long before we knew about the high level of genetic similarity, we knew that humans and chimps shared a similar morphology. Since our DNA largely determines organismal form, this high level of human-chimp genetic similarity could have been predicted without any knowledge of evolution, based upon observations of human and chimp morphological similarity alone. After all, similar designs usually imply a similar blueprint. Thus, this fusion evidence is not *independent* evidence for common ancestry. Yet as will be seen, the chromosomal fusion evidence simply strengthens the evidence for genetic similarity between chimps and humans. Since similarity could have been expected apart from Darwinism and common ancestry, similarities between organisms may just as easily be the result of functional requirements implemented via common design.

In *The Language of God*, Collins shows the highly similar karyotypes (i.e., sets of chromosomes) of chimps and humans.<sup>14</sup> Should this similarity

be surprising? As noted, morphological studies observe that humans and chimps are very similar in their body plan. Again, this similar morphology implies a similar genetic blueprint. Even if we had no concept of common ancestry, we would still *expect* that both species would share similar genetic blueprints, including their chromosomal scheme.

Now consider a hypothetical scenario where human chromosome 2 becomes unfused. In this case, humans would now have forty-eight chromosomes like chimpanzees. This hypothetical karyotype would closely resemble the chimp karyotype. Evidence for chromosomal fusion strengthens our already-strong knowledge that humans and chimps have a similar genetic makeup. But that's all the fusion evidence actually demonstrates.

### **A Thought Experiment: "The Doublefusers"**

As a final illustration of why the chromosomal fusion evidence does not logically provide special evidence for human-ape common ancestry, consider this hypothetical scenario. Assume that in 2015, two members of a small, isolated tribe of humans, who have never had contact with modern civilization, experience a second chromosomal fusion event. Within a few generations, this trait spreads throughout the small tribe until all members have twenty-two pairs of chromosomes (they remain fertile and normal in all other respects). We'll call the tribe the "*Doublefuser*" people. In 2100, war, famine, and sickness kill off the entire human race except for this remote tribe, and the human species experiences a genetic bottleneck. Many centuries later, the descendants of the *Doublefuser* tribe repopulate the earth and rediscover genetics and Darwinian evolution.

The technologically advanced *Doublefusers* examine their karyotype and proclaim, "*Wow, we humans have two pairs of fused chromosomes. Were we to unfuse these two pairs, our karyotype looks just like a chimp karyotype. These two fusion events show that we share common ancestry with apes.*"

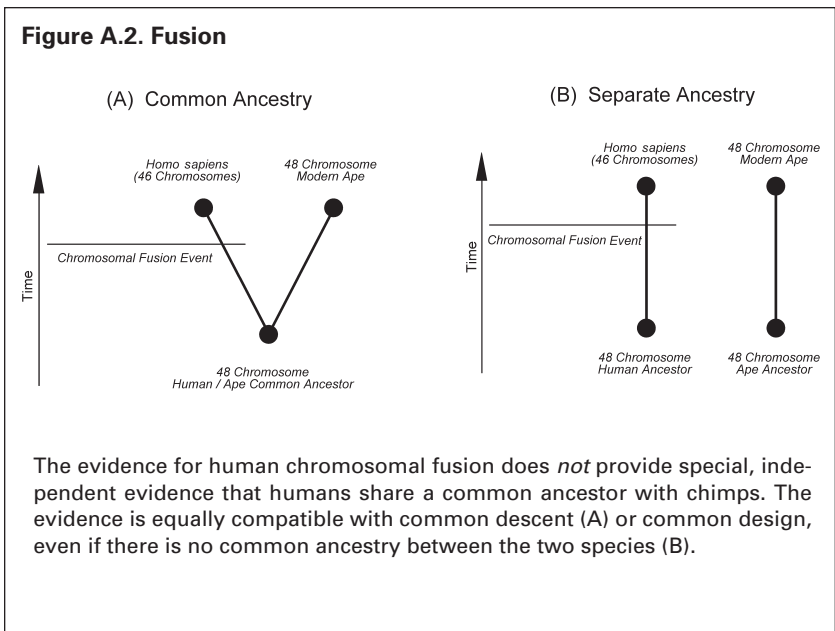
But of course, from our present vantage point, we understand the second pair of fused chromosomes in *Doublefusers* has nothing to do with chimp genetics and is far removed from any hypothetical common ancestor between humans and chimps: The second fusion event took place in 2015, and there's no logical reason why the second fusion event should cause them to infer common ancestry with apes. Yet modern Darwinists view our one pair of fused chromosomes precisely as the *Doublefusers* view

their two pairs of fused chromosomes. We know that the second fused chromosome in the *Doublefusers* logically provides no special evidence for chimp-human ancestry. Why should we assume the case must be any different with our one fused chromosome?

**Common Design or Common Descent Explain Fusion Evidence**

As the illustration demonstrates, the fusion evidence does not tell us whether human chromosomal fusion took place in a line that leads back to a common ancestor with chimps or in an independent line that was designed separately. What it demonstrates is that at one time our lineage had forty-eight chromosomes, similar to current ape genetic structure. As illustrated below, chromosomal fusion can exist in the human lineage without a human-ape common ancestor.

As seen in figure A.1, the evidence for human chromosomal fusion does *not* provide independent evidence that humans share a common ancestor with chimps. As seen in figure A.2, the evidence is equally compatible with common ancestry (A) and separate ancestry (B).



The chromosomal fusion evidence strengthens our knowledge that humans and chimps have similar genetics. But we could have anticipated these genetic similarities without evolution. And Collins himself admits that shared functional similarities between two organisms does little to assist in discriminating between common ancestry and common design with separate ancestry. Thus, the evidence for chromosomal fusion in humans provides no special evidence for common ancestry between humans and apes.

### **“Junk” DNA in Humans and Apes**

Intelligent design can explain functional similarities, but what about non-functional similarities? Like others before him, Collins argues that common ancestry should be inferred from segments of DNA that *appear* to be useless, since such segments are often found to be in the same places in human and chimp chromosomes.

At best, Collins’s “junk” DNA arguments explain dysfunction in the genetic text—biological gibberish. What he fails to explain is what generated the biological information in the first place. For this explanation, we must turn to intelligent design.

Intelligent design is primarily a historical science. It studies present-day causes and applies them to the historical record to *infer* the best explanation for natural phenomena. Intelligent design uses uniformitarian reasoning, based upon the principle that “the present is the key to the past.”<sup>15</sup> It starts with observations of intelligent agents to establish a cause-and-effect relationship between intelligence and the generation of certain types of information. William Dembski writes that “the defining feature of intelligent causes is their ability to create novel information and, in particular, specified complexity.”<sup>16</sup> Stephen Meyer explains that we are justified in inferring design in biology when we find specified complexity:

[W]e have repeated experience of rational and conscious agents—in particular ourselves—generating or causing increases in complex specified information, both in the form of sequence-specific lines of code and in the form of hierarchically arranged systems of parts. . . . Our experience-based knowledge of information-flow confirms that systems with large amounts of specified complexity (especially codes

and languages) invariably originate from an intelligent source from a mind or personal agent.<sup>17</sup>

Intelligent design accounts for the origin of new genetic information, because in our experience the type of specified and complex information found in human DNA *always* comes from intelligence. In contrast, Collins does not convincingly explain how new functional DNA first originated.

As molecular biologists learn more about DNA, they are continually testing—and refuting—the Darwinian hypothesis that DNA is mainly useless genetic “junk.” In the past, Darwinists loudly proclaimed that much of our DNA contained repetitive elements that are meaningless.<sup>18</sup> Darwinists have claimed this major portion of DNA is selfish or parasitic DNA that invades and becomes inserted into our genomes:<sup>19</sup> only eight years ago, Richard Dawkins specifically targeted repetitive DNA as undesignated “junk,” writing that “creationists might spend some earnest time speculating on why the Creator should bother to litter genomes with . . . junk tandem repeat DNA.”<sup>20</sup>

Collins similarly assumes that these stretches of DNA with a repeating sequence, called “ancient repetitive elements” (AREs), are nonfunctional “genetic flotsam and jetsam.”<sup>21</sup> Sounding much like Dawkins, Collins claims that “truncated” or “decapitated” repetitive elements “presen[t] an overwhelming challenge to those who hold to the idea that all species were created *ex nihilo*.”<sup>22</sup> Collins writes, “Unless one is willing to take the position that God has placed these decapitated AREs in these precise positions to confuse and mislead us, the conclusion of a common ancestor for humans and mice is virtually inescapable.”<sup>23</sup>

While Collins casts his challenge in theological terms, a scientific assessment is possible. Collins is wrong to make an argument from ignorance and assume that AREs (or “truncated AREs”) have no function, merely because no function is currently known. In 2002, evolutionary biologist Richard Sternberg surveyed the literature and found extensive evidence for function in AREs. Sternberg’s article concluded that “the selfish DNA narrative and allied frameworks must join the other ‘icons’ of neo-Darwinian evolutionary theory that, despite their variance with empirical evidence, nevertheless persist in the literature.”<sup>24</sup> Reprinted from Sternberg’s paper, known genomic/epigenetic roles of REs include:

## Appendix

- satellite repeats forming higher-order nuclear structures;
- satellite repeats forming centromeres;
- satellite repeats and other REs involved in chromatin condensation;
- telomeric tandem repeats and LINE elements;
- subtelomeric nuclear positioning/chromatin boundary elements;
- non-TE interspersed chromatin boundary elements;
- short, interspersed nuclear elements or SINEs as nucleation centers for methylation;
- SINEs as chromatin boundary/insulator elements;
- SINEs involved in cell proliferation;
- SINEs involved in cellular stress responses;
- SINEs involved in translation (may be connected to stress response);
- SINEs involved in binding cohesion to chromosomes; and
- LINEs involved in DNA repair.

Other genetic research continues to uncover functions for allegedly functionless types of repetitive DNA, including SINE,<sup>25</sup> LINE,<sup>26</sup> and ALU elements.<sup>27</sup> Sternberg, along with leading geneticist James A. Shapiro, concludes elsewhere that “one day, we will think of what used to be called ‘junk DNA’ as a critical component of truly ‘expert’ cellular control regimes.”<sup>28</sup>

Collins asserts that truncated AREs are “junk.” But geneticists are constantly disproving the assumption that non-coding DNA is “junk.” For example, a 2003 *Scientific American* article, “The Unseen Genome: Gems Among the Junk,” explains that types of non-coding DNA were “long ago written off as irrelevant because they yield no proteins.”<sup>29</sup> Though written from an evolutionary perspective, the article further says these introns “were immediately assumed to be evolutionary junk”<sup>30</sup> but admits that assumption was hasty. What’s more, the author writes that this failure to recognize introns as functional is possibly “one of the biggest mistakes in the history of molecular biology.”<sup>31</sup> Similarly, a paper from the *Annals of the New York Academy of Sciences* argues that “neo-Darwinian ‘narratives’ have been the primary obstacle to elucidating the effects of these enigmatic components of chromosomes,” so “a new conceptual framework is needed.”<sup>32</sup>

Indeed, the whole notion of “junk” DNA is quickly unraveling, as the *Washington Post* reported: “A project involving hundreds of scientists in 11 countries and detailed in 29 papers” discovered that “the vast majority of the 3 billion ‘letters’ of the human genetic code are busily toiling at an array of previously invisible tasks.”<sup>33</sup> Collins should be wary of repeating the mistaken assumption that types of non-coding DNA have no function, especially when multiple types of functions have been found for AREs.

In another “junk” DNA argument, Collins writes that human pseudo-gene caspase-12 is functionless and asks, “why would God have gone to the trouble of inserting such a nonfunctional gene in this precise location?”<sup>34</sup> But Collins acknowledges that the caspase-12 gene produces a full-fledged protein in chimps, so this is not a case where humans share a nonfunctional stretch of DNA with another species. There is no evidence that humans inherited the gene’s nonfunctional state from a common ancestor with chimps, lending little support to common descent. In fact, 28 percent of people in sub-Saharan Africa have a functioning copy of the caspase-12 gene, as do lower percentages in some other human populations.<sup>35</sup> Collins ignores the obvious explanation that caspase-12 was originally designed to be functional in humans but was rendered nonfunctional by a mutation in most human populations in the very recent past. Like the evidence for chromosomal fusion, this pseudogene tells a story of an event that occurred uniquely within the human lineage and does not necessarily say anything about whether humans share an ancestor with apes.

Functions for other pseudogenes have already been discovered.<sup>36</sup> As with the AREs, why, then, should we assume that even the allegedly broken copy of caspase-12 is functionless “junk”? One study suggested that even in humans with the “premature” stop codon, which is said to turn caspase-12 into a pseudogene, it still produces a “CARD-only protein,”<sup>37</sup> which can be a type of functional protein in humans.<sup>38</sup> The study suggests that the similarity between human caspase-12 and other CARD-only proteins could provide evidence that human caspase-12 interacts in some biological pathways, and it recommends that scientists study the human caspase-12 to determine what it does:

Since human pseudo-caspase-12 is structurally comparable to ICE-BERG and COP/Pseudo-ICE [CARD-only proteins], it would be interesting to study its involvement in similar pathways.<sup>39</sup>

While the study suggests searching out a function for human caspase-12, Collins makes an argument from ignorance. He would advise that we assume the pseudogene does nothing and stop searching for a function. As in the case of introns, this could be a false Darwinian assumption that hinders research.

It would be more prudent for Collins to acknowledge that functions are already known for many AREs and pseudogenes and to adopt a wait-and-see approach for those types of DNA (like truncated AREs or some other pseudogenes) whose functions we do not yet understand. The history of genetics teaches us not to assume that poorly understood types of DNA are merely functionless junk. And if they aren't functionless junk, this may be another instance where, in Collins's own words, a designer could have "used successful design principles over and over again."<sup>40</sup>

### **Non-Coding DNA Sequences Have More Differences Than Protein-Coding Sequences of DNA**

Despite the rapidly accumulating evidence that non-coding DNA has function, Collins and other Darwinists continue to assume that much non-coding DNA really is "junk."<sup>41</sup> Collins says that "Darwin's theory predicts that mutations that do not affect function (namely, those located in 'junk DNA') will accumulate" more rapidly than those in "the coding regions of the genes."<sup>42</sup> He cites a greater level of differences among species' non-coding DNA than among their protein-coding DNA as evidence that the non-coding DNA is "junk." He assumes that the large differences between non-coding DNA stem from the more-rapid accumulation of random mutations in those allegedly "junk"-filled regions.

Collins's view ignores the hypothesis that non-coding DNA *is* performing a key function, such as controlling gene expression, such that the differences in non-coding DNA have a large influence on organismal development. If this is the case, then the Darwinian "junk" DNA viewpoint has again slowed the progress of science.

In a recent *Time* magazine article, some skeptics of "junk" DNA make precisely this point.<sup>43</sup> They contend that Collins's observation simply demonstrates that many of the genetic differences responsible for physical differences between organisms lie in their non-coding DNA. Because the human genome project revealed that humans have so few genes, the article



explains, some scientists have reasoned that the differences between species must be controlled not by genes but by stretches of DNA that *do not code for proteins*. To explain this point, the *Time* article quotes evolutionist Owen Lovejoy:

This shockingly small number made it clear to scientists that genes alone don't dictate the differences between species; the changes, they now know, also depend on molecular switches that tell genes when and where to turn on and off. "Take the genes involved in creating the hand, the penis and the vertebrae," says Lovejoy. "These share some of the same structural genes. The pelvis is another example. Humans have a radically different pelvis from that of apes. It's like having the blueprints for two different brick houses. The bricks are the same, but the results are very different."<sup>44</sup>

Indeed, a recent article in *Nature* titled, "It's the junk that makes us human," argues this same point:

Anyone who has ever put together self-assembly furniture knows that having the right parts is important, but what you do with them can make or break the project. The same seems to be true of the vast amounts of DNA in an organism's genome that used to be labelled as junk. Studies now indicate that this DNA may be responsible for the signals that were crucial for human evolution, directing the various components of our genome to work differently from the way they do in other organisms.

The findings seem to bolster a 30-year-old hypothesis that gene regulation—not the creation of new genes—has moulded the traits that make us unique.

The latest work looks for regions of the genome that have changed rapidly in human evolution, based on the theory that they are most likely to have shaped our differences from other animals. But instead of hunting for rapidly evolving DNA in genes, researchers are starting to look at non-coding DNA—stretches of DNA that don't encode proteins.<sup>45</sup>

Thus, the higher level of differences between non-coding DNA is not evidence that it is merely "junk" that is accumulating random mutations at a higher rate than protein-coding DNA. Rather, the large differences

show that non-coding DNA has functionality that controls gene expression, which is important for determining the physical characteristics of an organism. This model accounts for the surprisingly low number of genes (i.e., segments of coding DNA) in organisms because it shows that building an organism requires more than merely the gene-coding segments of DNA. This hypothesis easily accounts for the raw data that Collins presents.

In *The Language of God*, Collins provides a chart with the caption, “Likelihood of Finding a Similar DNA Sequence in the Genome of Other Organisms, Starting with a Human DNA Sequence.”<sup>46</sup> He then provides statistics that compare the similarities between human DNA sequences and DNA sequences of other species. Two types of DNA sequences are compared: those that code for proteins, and those that do not code for proteins (called “non-coding” sequences). In his chart, protein-coding DNA sequences between humans and other species are more similar than are the non-coding DNA of humans and other species.

Collins reports that some protein-coding DNA sequences between humans and chimps are 100 percent identical. But non-coding DNA sequences between humans and chimps are apparently only 98 percent similar. Following the same pattern, Collins reports that human protein-coding DNA is 99 percent similar to that of dogs or mice. But human non-coding DNA is only 52 percent and 40 percent similar to the non-coding DNA of dogs and mice, respectively. The differences are most pronounced with invertebrates. Human protein-coding DNA is 60 percent similar to fruit flies. But here, non-protein coding DNA is reportedly about 0 percent similar to that of fruit flies. Collins assumes these non-coding sequences are junk, because his chart labels them, “Random DNA Segment[s] Between Genes.”

Collins uses this data to assert that the reason species have a greater degree of difference between their non-coding DNA than their coding DNA is because non-coding DNA is junk that accumulates mutations at a higher rate. But if we do not make the questionable evolutionary assumption that non-coding DNA is junk, then this data simply suggests that physical differences between different species are not largely controlled by protein-coding DNA but are rather controlled by the non-coding DNA. Yet Collins considers these vital non-coding differences to be the result

of “random DNA segment[s] between genes.”<sup>47</sup> Large differences exist in non-coding DNA among widely different species because the non-coding DNA may be largely responsible for differences between species.

Now that differences in non-coding DNA are known to control gene expression (which influences an organism’s growth and development), we cannot assume, with Collins, that the differences among “junk” DNA in different species are the result of random mutations. Rather, such differences may be better explained as designed differences tailored precisely to encode the developmental traits among species. In fact, the conclusion that non-coding DNA controls gene expression and helps determine physical characteristics is precisely what intelligent design would predict. ID would not expect that large sections of DNA be mere junk. Collins’s argument reveals why the Darwinian “junk” DNA assumption might hinder scientific research.

### **Mutations That Allegedly Caused the Evolution of Human Cognition and Language**

Two arguments made by Collins are repeated in the aforementioned *Time* magazine article on chimp and human similarities.<sup>48</sup> Both appear to invoke extravagant and highly unlikely macromutations to account for the origin of major aspects of human intellect.

#### **A Jaw-Dropping Theory**

First, Collins looks at the MYH16 gene, which supposedly mutated into a pseudogene in humans.<sup>49</sup> Since MYH16 affects the production of myosin in jaw muscles, a protein that effectively increases muscle strength, it is said that the alleged loss of this gene caused jaw muscles to be weaker. Some have hypothesized this loss in jaw-muscle strength allowed the human braincase to grow larger, causing an increase in human intelligence.

This is a nice story, but does it make sense? Around the same time this research was first reported, leading paleoanthropologist Bernard Wood explained why simply identifying the effects of a mutation does not imply that we have an accurate evolutionary story:

The mutation would have reduced the Darwinian fitness of those individuals. . . . It only would’ve become fixed if it coincided with

mutations that reduced tooth size, jaw size and increased brain size. What are the chances of that?<sup>50</sup>

But if we accept the story that human brains enlarged due to this unlikely mutation, the implication is that this explains human intellectual evolution. But the well-known paleoanthropologist Ian Tattersal observes that simply enlarging the brain does very little to explain the evolution of human cognition:

[T]he arrival of the modern cognitive capacity did not simply involve adding just a bit more neural material, that last little bit of extra brain size that pushed us over the brink. Still less did it involve adding new brain structures, for basic brain design remains remarkably uniform among all the higher primates.<sup>51</sup>

### **A Miracle Macromutation Theory of the Evolution of Human Cognition**

So how did our higher brain capacities evolve, genetically speaking? Again Collins thinks he has an answer. As his second argument, he discusses what is essentially a highly improbable miracle mutation in which two changes in our FOXP2 gene created some of our major linguistic abilities.<sup>52</sup> The *Time* article makes an even bolder claim, asserting that the two mutations could have caused “the emergence of all aspects of human speech, from a baby’s first words to a Robin Williams monologue.”<sup>53</sup> Such an optimistic line of reasoning strains credulity. The origin of human speech would have required many changes that make up a suite of interdependent complex traits. Two leading evolutionists writing in a prominent text on primate origins explain that human language could not evolve in an abrupt manner, genetically speaking:

How could we move from communication systems in nonhuman primates to human language in a manner consistent with evolutionary principles? Arguments that humans are fundamentally different from nonhuman animals either set the stage for creationist explanations or simply avoid the attempt to develop a persuasive evolutionary argument. Bickerton’s proposal of a single-gene mutation is, I think, too simplistic. Too many factors are involved in language learning—

production, perception, comprehension, syntax, usage, symbols, cognition—for language to be the result of a single mutation event.<sup>54</sup>

Humans are quite different because they possess language, which underlies every major intellectual achievement of humanity. This discontinuity theory is implausible because evolution cannot proceed by inspired jumps, only by accretion of beneficial variants of what went before.<sup>55</sup>

These authors are correct to reject the “single mutation event” hypothesis—and they would be justified in doing the same for two mutation events because human language is vastly more complex than the form of communication found even in our allegedly closest relative, the chimpanzee. Rather than supporting Collins’s “single event hypothesis,” these authors would argue that human intellectual capacities are different only in degree from those of other primates, and thus could have evolved in a slow, step-by-step fashion. But this view has its own problems. Ian Tattersal describes the intellectual gulf between humans and other species, saying, “Human language is governed by its structure, which admits endless possibilities; ape vocalization is governed by its content, which is inherently limited by its mode of expression.”<sup>56</sup> This miracle macromutation theory of the origin of language is attractive to evolutionists because all humans appear to be hardwired for language.<sup>57</sup> One Darwinist, Elizabeth Bates, suggests that this leaves two unviable options for evolutionists:

If the basic structural principles of language cannot be learned (bottom up) or derived (top down), there are only two possible explanations for their existence: either Universal Grammar was endowed to us directly by the Creator, or else our species has undergone a mutation of unprecedented magnitude, a cognitive equivalent of the Big Bang.<sup>58</sup>

What protoform can we possibly envision that could have given birth to constraints on the extraction of noun phrases from an embedded clause? What could it conceivably mean for an organism to possess half a symbol or three quarters of a rule? . . . monadic symbols, on a yes-or-no basis—a process that cries out for a Creationist explanation.<sup>59</sup>

Some Darwinists, such as Steven Pinker, think language can evolve piecemeal from forms of protolanguage. But other Darwinists like Tattersal disagree because “language is a unique aptitude that doesn’t seem to have emerged from protolanguage.”<sup>60</sup> Those who follow Tattersal’s view accept the integrated complexity of language and may prefer the miracle macro-mutation hypothesis. But as we’ve already seen, that explanation doesn’t jibe with slow, gradual, step-by-step Darwinian modes of change.

Because of this conundrum, Tattersal is forced to argue that human language and cognition evolved by an accidental, indirect route, where key steps in our evolution did not even provide the types of cognitive advantages humans currently enjoy. Such a process of indirect and purely accidental route of evolution is called “exaptation,” and Tattersal thus argues that we have “an exapted brain, equipped since who knows when with a neglected potential for symbolic thoughts.” According to Tattersal, language evolved after a change that was “minor in genetic terms, and probably had nothing whatever to do with adaptation in the classical sense.”<sup>61</sup> In other words, lower primate brains just happened to have all the tools ready for complex human thought and language, even though they weren’t being used for such, and a small random mutation in our hominid ancestors that wasn’t even adaptive caused human language. This option, too, seems incredible.

Perhaps language presents the type of complex feature contingent on many variables that Noam Chomsky as well as “some of his fiercest opponents” believe is “incompatible with the modern Darwinian theory of evolution, in which complex biological systems arise by the gradual accumulation over generation of random genetic mutations that enhance reproductive success.”<sup>62</sup> Would Collins consider this option?

## Conclusion

Francis Collins’s position as both a Christian and a Darwinian scientist enables him to make valuable contributions to the debate over evolution. However, his Darwinian arguments in favor of human-ape common ancestry are simply unconvincing:

- Collins argues that chromosomal fusion in humans is strong evidence for the hypothesis of human-ape common ancestry. Yet this

fusion event took place entirely within the human line after our alleged split with the chimpanzee line and by itself does not tell us whether the human line extends back to share a common ancestor with apes. At best, this evidence demonstrates that humans and chimps share similar genetics—something we already knew without evolutionary biology. Such similarities are just as easily explained by common design.

- Collins assumes that some non-coding DNA is functionless “junk.” That such an eminent geneticist as Collins would make so dubious an assumption, given its well-documented history of failure, makes clear how entrenched the “junk” DNA mind-set is within the Darwinian scientific community.
- Collins argues that the larger degree of difference in non-coding DNA compared to protein-coding DNA among various species indicates that non-coding DNA is functionless junk that has accumulated many random mutations. By extension, Collins suggests this is evidence of common ancestry and a Darwinian past. Yet his assumption of nonfunctionality is increasingly betrayed by data showing that non-coding DNA controls gene expression, which is precisely what helps cause differences in the physical characteristics of different species.
- Collins suggests that the evolution of human cognition and language could be explained by a few small mutations. In reality, the origin of human intellect would have required a complex series of coordinate changes that challenge a Darwinian explanation.

discrimination in reference to teaching intelligent design. Questions of freedom of religion or establishment of religion should be avoided.

186. The errors of attention attracted by having church groups seek to influence legislation should be avoided. Sponsors should restrict themselves to concerns of better science education and academic freedom for students.

### **Appendix: A Reply to Francis Collins’s Darwinian Arguments for Common Ancestry of Apes and Humans**

1. Francis Collins, *The Language of God: A Scientist Presents Evidence for Belief* (New York: Free Press, 2006), 199.
2. For example, see David Van Biema, “God vs. Science,” *Time* (November 13, 2006): 48–55; and David Van Biema, “The Discover Interview: Francis Collins,” *Discover* (February 2007): 44–47.
3. Two reviews of Collins’s book concur that Collins does a good job of explaining and supporting arguments for intelligent design in cosmology, but then inconsistently rejects intelligent design in biology. See Logan Paul Gage, “Best Explanations,” *American Spectator* (October 1, 2006), at [discovery.org/scripts/viewDB/index.php?command=view&id=3749](http://discovery.org/scripts/viewDB/index.php?command=view&id=3749). See also Jonathan Witt, “Book Review: Random Acts of Design: Francis Collins Sees Evidence That God Made the Cosmos—But Life Is Another Matter,” *Touchstone* (November 9, 2006), at [touchstonemag.com/archives/article.php?id=19-08-032-f](http://touchstonemag.com/archives/article.php?id=19-08-032-f).
4. See Michael J. Behe, *The Edge of Evolution: The Search for the Limits of Darwinism* (New York: Free Press, 2007).
5. See Jonathan Wells, “Darwin’s Tree of Life,” in *Icons of Evolution* (Washington, DC: Regnery, 2000).
6. W. Ford Doolittle, “Phylogenetic Classification and the Universal Tree,” *Science* 284 (June 25, 1999): 2124–28.
7. W. Ford Doolittle, “Uprooting the Tree of Life,” *Scientific American* (February 2000): 90–95.
8. Carl Woese, “The Universal Ancestor,” *Proceedings of the National Academy of Sciences USA* 95 (June 1998): 6854–9859.
9. Antonis Rokas, Dirk Krüger, Sean B. Carroll, “Animal Evolution and the Molecular Signature of Radiations Compressed in Time,” *Science* 310 (December 23, 2005): 1933–38.
10. Stephen C. Meyer, Marcus Ross, Paul Nelson, and Paul Chien, “The Cambrian Explosion: Biology’s Big Bang,” in *Darwinism, Design, and Public Education*, ed. John Angus Campbell and Stephen C. Meyer (East Lansing, MI: Michigan State University Press, 2003).
11. Collins, *The Language of God*, 134.
12. The original paper on this is Jorge J. Yunis, Jeffrey R. Sawyer, and Kelly Dunham, “The Striking Resemblance of High-Resolution G-banded Chromosomes of Man and Chimpanzee,” *Science* 208 (1980): 1145–48. A



- follow-up paper that is widely cited is: Jorge J. Yunis and Om Prakash, "The Origin of Man: A Chromosomal Pictorial Legacy," *Science* 215 (1982): 1525–30. Information from these papers is frequently found in textbooks. For example: Gerald J. Stine, *The New Human Genetics* (Dubuque, IA: Wm. C. Brown, 1989), 313.
13. Nearly all blueprints for houses include sketches of doors, walls, ventilation, etc. This does not mean that one blueprint was necessarily taken from another. Rather, the functional requirements of houses require that houses have ways to enter and exit, structural support, and ventilation.
  14. Collins, *The Language of God*, 137.
  15. Stephen C. Meyer, "The Cambrian Information Explosion," in *Debating Design*, ed. Michael Ruse and William Dembski (New York: Cambridge University Press, 2004).
  16. William A. Dembski, *No Free Lunch* (Lanham, MD: Rowman & Littlefield, 2002), xiv.
  17. Stephen C. Meyer, "The Origin of Biological Information and the Higher Taxonomic Categories," *Proceedings of the Biological Society of Washington* 117.2 (2004): 213–39.
  18. Jonathan Marks, *What It Means to Be 98% Chimpanzee: Apes, People, and Their Genes* (Berkeley, CA: University of California Press, 2002), 34.
  19. James A. Shapiro and Richard Sternberg, "Why Repetitive DNA Is Essential to Genome Function," *Biological Review* 80 (2005): 227–50.
  20. Richard Dawkins, "The Information Challenge," *The Skeptic* 18.4 (December 1998), at [simonyi.ox.ac.uk/dawkins/WorldOfDawkins-archive/Dawkins/Work/Articles/1998-12-04infochallenge](http://simonyi.ox.ac.uk/dawkins/WorldOfDawkins-archive/Dawkins/Work/Articles/1998-12-04infochallenge).
  21. Collins, *The Language of God*, 136.
  22. *Ibid.*, 137.
  23. *Ibid.*, 136–37.
  24. Richard Sternberg, "On the Roles of Repetitive DNA Elements in the Context of a Unified Genomic–Epigenetic System," *Annals of the New York Academy of Sciences* 981 (2002): 154–88.
  25. *Ibid.*
  26. Tammy A. Morrish et al., "DNA Repair Mediated by Endonuclease-independent LINE-1 Retrotransposition," *Nature Genetics* 31.2 (June 2002): 159–65.
  27. Galit Lev-Maor, Rotem Sorek, Noam Shomron, and Gil Ast, "The Birth of an Alternatively Spliced Exon: 3' Splice-site Selection in Alu Exons," *Science* 300.5623 (May 23, 2003): 1288–91; and Wojciech Makalowski, "Not Junk After All," *Science* 300.5623 (May 23, 2003): 1246–47.
  28. Richard Sternberg and James A. Shapiro, "How Repeated Retroelements Format Genome Function," *Cytogenetic and Genome Research* 110 (2005): 108–16.
  29. W. Wayt Gibbs, "The Unseen Genome: Gems Among the Junk," *Scientific American* (November 2003): 46–53, emphasis added.

## Notes

30. Ibid.
31. Ibid.
32. Sternberg, "On the Roles of Repetitive DNA Elements," 154–88.
33. Rick Weiss, "Intricate Toiling Found in Nooks of DNA Once Believed to Stand Idle," *Washington Post* (June 14, 2007), A01.
34. Collins, *The Language of God*, 139.
35. Yali Xue et al., "Spread of an Inactive Form of Caspase-12 in Humans Is Due to Recent Positive Selection," *American Journal of Human Genetics* 78 (April 2006): 659–70.
36. Shinji Hirotsune et al., "An Expressed Pseudogene Regulates the Messenger-RNA Stability of Its Homologous Coding Gene," *Nature* 423 (May 1, 2003): 91–96.
37. M. Lamkanfi, M. Kalai, and P. Vandenabeele, "Caspase-12: An Overview," *Cell Death and Differentiation* 11 (2004): 365–68.
38. Sug Hyung Lee, Christian Stehlik, and John C. Reed, "COP, a Caspase Recruitment Domain-containing Protein and Inhibitor of Caspase-1 Activation Processing," *Journal of Biological Chemistry* 276.37 (September 14, 2001): 34495–500.
39. Lamkanfi, Kalai, and Vandenabeele, "Caspase-12: An Overview," 365–68.
40. Collins, *The Language of God*, 134.
41. We recognize that Collins at certain points notes that little is known about "junk" DNA and claims it requires hubris to call it "junk" DNA. Collins, *The Language of God*, 111. Nonetheless, Collins ignores his own advice. He assumes that various types of DNA have no function.
42. Ibid., 129–30.
43. Michael D. Lemonick and Andrea Dorfman, "What Makes Us Different?" *Time* (October 1, 2006), at [time.com/time/magazine/article/0,9171,1541283,00](http://time.com/time/magazine/article/0,9171,1541283,00).
44. Ibid.
45. Erika Check, "It's the Junk That Makes Us Human," *Nature* 444 (November 9, 2006): 130.
46. Collins, *The Language of God*, 127.
47. Ibid.
48. Lemonick and Dorfman, "What Makes Us Different?"
49. Collins, *The Language of God*, 139.
50. Bernard Wood, quoted in Joseph B. Verrengia, "Gene Mutation Said Linked to Evolution," Associated Press, taken from the *San Diego Union Tribune*, March 24, 2004.
51. Ian Tattersal, *The Monkey In the Mirror: Essays on the Science of What Makes Us Human* (Orlando, FL: Harcourt, 2002), 160.
52. See Collins, *The Language of God*, 139–41.
53. Lemonick and Dorfman, "What Makes Us Different?"

54. Charles T. Snowdon, "From Primate Communication to Human Language," 224, in *Tree of Origin: What Primate Behavior Can Tell Us About Human Social Evolution*, ed. Frans B. M. de Waal (Cambridge, MA: Harvard University Press, 2001).
55. Richard W. Byrne, "Social and Technical Forms of Primate Intelligence," in deWaal, ed., *Tree of Origin*, 148–49.
56. Tattersal, *The Monkey in the Mirror*, 158.
57. See Carl Zimmer, *Evolution: The Triumph of an Idea* (New York: Harper Collins, 2001), 286–89.
58. Elizabeth Bates quoted in Steven Pinker, *The Language Instinct: How the Mind Creates Language* (New York: Harper Perennial, 1994), 350.
59. *Ibid.*, 377.
60. Tattersal, *The Monkey in the Mirror*, 160.
61. *Ibid.*, 162.
62. Noam Chomsky's belief as described in Pinker, *The Language Instinct*, 341. Pinker rejects Chomsky's view and believes that language can evolve in a Darwinian fashion. Readers are referred to chapter 11 of Pinker's book for details.