flip and see if such a statistical model can explain the observed chromosomal organization. It assumes that the transcription direction for each gene has a probability of 0.5 and that the probabilities are independent of each other. This flip-of-the-coin model would predict that the average number of genes on a string should lie near 2 per string. That is not what is observed in chromosomal organization. In Table 1 we list the number of genes per string (n/s) and then the 3 standard deviation range for each entry. When we compare the string lengths observed with the expected range for the coin-flip model, we see significant deviations from that in Table 1 among the archaea and bacteria. If the string lengths are consistent with this model, the n/s value has approximately a 99% chance of falling between the 3-sigma values. The clear conclusion is that this random model totally fails to account for the bacterial data. However, the gene strings of the "higher" eukaryotes, like yeast and *C. elegans*, are clearly more randomized when measured against this model. Indeed, they seem to lie within the statistically predicted values given by this flip-of-the-coin model, or nearly so. This is a surprising result when the above statements are taken into account. Moreover, a slight modification of this flip-of-the-coin model yields a very compelling fit for each of the three eukaryotes, a fit that is good enough to pass a demanding goodness-of-fit test. The standard flip-of-the-coin model assigns p = 0.5 as the probability that a given space between adjacent chromosomes is a transition point. The modification for *D. melanogaster* is to assign p = 0.541 (the very same value of p for all 19 scaffolds!). Comparable, excellent fits are possible for yeast, with p = 0.530 (the same value of p for all 16 chromosomes!), and for *C. elegans*, with p = 0.463 (the same value of p for all six chromosomes!). The quality of the fits under p = 0.5 are not good enough to pass the demanding statistical test referred to above. We will return to the philosophical implications of this below.

Does this mean that the bacteria are incompatible with random chance when their gene strings are examined? Two other models were tested attempting to determine if a stochastic model could reasonably match the observed gene string length.

Model 2 starts with the observed data for each species—the number of genes and the number of strings. Labeling the space between two strings as a transition point, the number of transition points is equal to the number of strings (except when there is only one string—and no transition point). Model 2 next distributes the transition points randomly among the n available spaces between the genes. Then the distribution of the resulting string lengths is compared with the actual string lengths of the chromosome. In turn, the distribution of string lengths

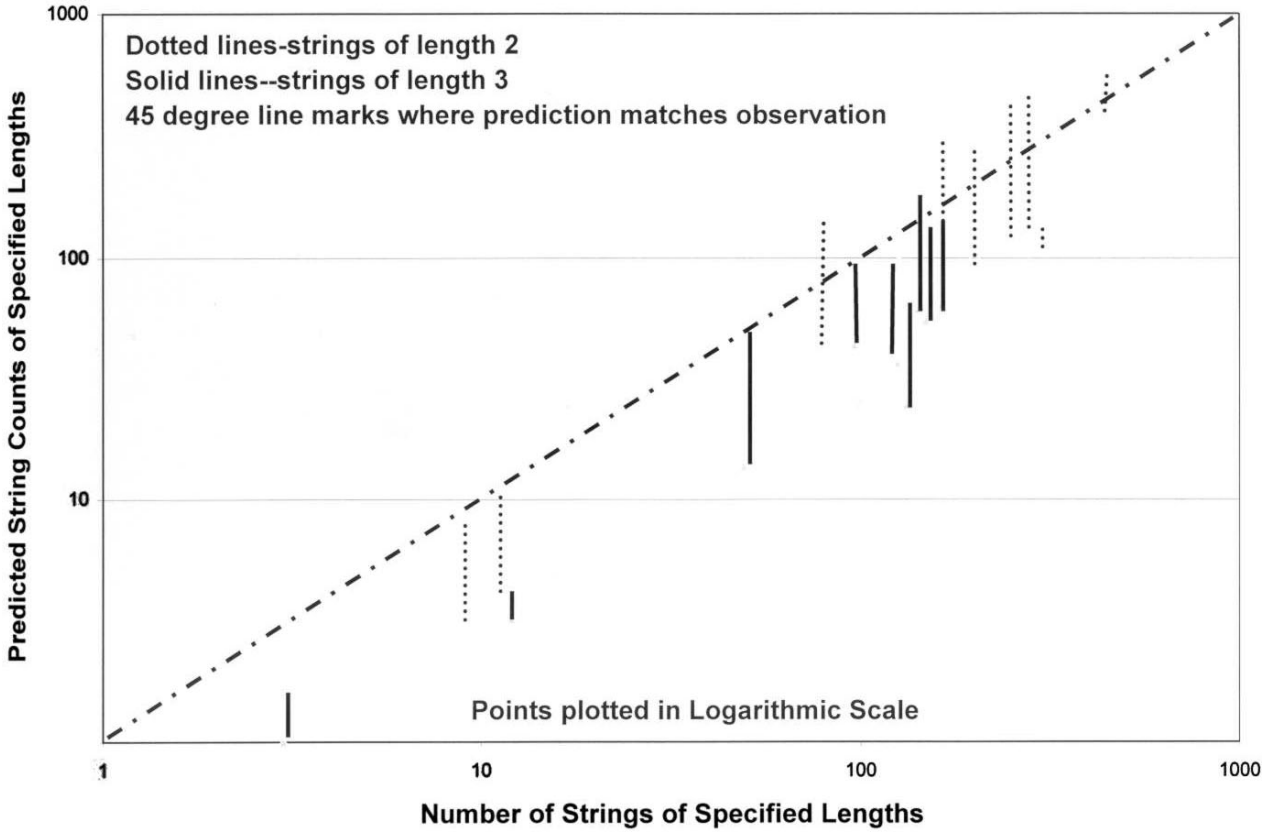

Figure 2. The results of Model 3.

Bacteria appear to have a genetic structure that is decidedly nonrandom. And one might be tempted to conclude that life indeed is a nonrandomly organized phenomenon. … But this does not explain why the more complex eukaryotes studied here fit Model 1 (the flip-of-the-coin model) so well, …

is compared with that in the genome. This could be called the slice-and-dice model where the sequence of genes is randomly cut and every other segment inverted. This model underestimates the actual number of strings of length 1 a bit, estimates the strings of length 2 approximately correctly, and overestimates the number of strings of length 3 a bit, and fails to account for the extraordinary length of the longest strings found in the chromosomes. There is more variability in the string lengths of bacteria than can be accounted for by randomness described by Model 2.

Model 3 assumes that the orientations of the genes (and hence the length of each gene string) correspond to the signs of n correlated (mean zero, variance one) Gaussian random variables. Placing reasonable mathematical constraints on the correlations leads to a set of three equations with four unknowns. The values of these unknowns precisely determine the distribution of the string lengths. One can solve these equations numerically to obtain a range of solutions, and, thus, a set of possible string-length distributions. It is observed in Figure 10 of Hutchison, et al. and Figure 2 in this article that these fit the observed data well.

Implications of this Work

The most obvious implication is that bacteria appear to have a genetic structure that is decidedly nonrandom. And one might be tempted to conclude that life indeed is a nonrandomly organized phenomenon. This certainly fits the preconceptions of many Christian apologists and laity who see randomness as a threat to a particular theological position. Many apologists claim that order found in biology is evidence of God's design.[14] But this does not explain why the more complex eukaryotes studied here fit Model 1 (the flip-of-the-coin model) so well, and a slight modification extremely well. This goes against the common claim by apologists that "the information required for large-scale evolution cannot come from random variations"[15] and "… the integrated complexity of developmental programs [cannot] plausibly be attributed to chance."[16]

Why is it that *C. elegans*, a nematode that is more complex than bacteria, possesses a genetic structure that appears more randomized than the much simpler bacteria and archaea? *C. elegans* has a nervous system, muscle cells, a hypodermis, an excretory system, and a specialized reproductive system. As we saw above, the bacteria have the genes for entire biochemical systems located in the same string; from the yeast to the *C. elegans* and on up to humans (whose incomplete and early genome information has been examined by the authors), there is no such order (imposed by need), and the gene's transcription direction is free to be mutated randomly. It is hard to avoid the conclusion that the more complex organism is more "randomized."

The data we present here has implications to the common belief that random mutations and selection cannot improve a system. Such views are illustrated by that of Davis and Kenyon who said:

> A mutation in a coding gene, then, can be looked at as a random change in functional information. As a unit of functional information in the cell, a coding gene is much like a word (a unit of meaningful information) in a book. What do you think would happen if we randomly changed the letters in some of the words in this book? Would the book be improved? On the contrary, it is probable that random changes in the words of this book would decrease rather than increase the meaningful information they carry.[17]

The mathematically more randomized state of the eukaryote gene strings we examined is not quite the equivalent of randomly changing the letters in this article, but it is the equivalent of randomly reversing the direction in which the words are read. While the reading of the sentence, "Information is hurt by random reversal of the word directions" is made more difficult by laying it out as "noitamrofni si truh yb modnar lasrever fo eht drow snoitcerid" the same procedure applied to genes and gene systems seems to make for more complex beings—yeast, nematodes, fruit flies, and humans. This clearly shows that one ought not draw the analogy between words and genetic systems too closely. Words are not genetic systems and genetic systems are not words.

For those who truly understand information theory, the above results should not be

surprising. Hubert Yockey has stated that it is mathematically fundamentally impossible to tell the difference between a random sequence and one that codes for an organism![18] Indeed, the higher the information content of a sequence the more random the sequence will appear from a mathematical point of view. This is what we see with the gene strings of the higher organisms.

The ability of the modified coin-flip model to fit the observed gene string patterns seen in living systems calls into question the efficacy of William Dembski's methodology for determining design. Dembski wrote:

> Even so, complexity (or improbability) isn't enough to eliminate chance and establish design. If I flip a coin 1,000 times, I'll participate in a highly complex (i.e., highly improbable) event. Indeed, the sequence I end up flipping will be one in a trillion trillion trillion …, where the ellipsis needs 22 more "trillions." This sequence of coin tosses won't, however, trigger a design inference. Though complex, this sequence won't exhibit a suitable pattern. Contrast this with the previous sequence representing prime numbers from 2 to 101. Not only is this sequence complex, but it also embodies a suitable pattern. The SETI researcher who in the movie *Contact* discovered this sequence put it this way: "This isn't noise, this has structure."[18]

Dembski, like us, wants to conclude that living systems are designed. But Dembski's methodology for determining design, by his own admission, excludes anything that has the structure of a coin-flip model, which is exactly what we find in the patterns of the gene strings. Are we to conclude from this that the cells are not designed? No! What we can conclude is that Dembski's model is inadequate to the task he intends. Once again, as Yockey notes, organized sequences will appear the same as randomly generated sequences. As an example, consider the sequence "yjrvsyonstrfdjoty" which is a Caesar substitution cipher for "thecatintheredshirt." Both sequences contain the same meaning and semantical information. The only reason the latter, decrypted sequence does not look randomized is because of years of training in how to read letters in that order.

Conclusions

We have presented evidence for both random and nonrandom features of the chromosomal structure. The asymmetries in the gene directionality and the bacterial deviations from the predictions of our models must be due to nonrandom forces and mechanisms required for life to exist. But at the same time, there is already much evidence that the chromosomal organization of the eukaryotes is nearly consistent with a simple flip-of-the-coin model and show more randomization than do the bacteria and archaea.

It must be concluded from our study that many of the apologetical statements about living systems do not find unequivocal empirical support. The observation that the chromosomes of more advanced animals appear more random does not support the concept that information and randomness are incompatible. The higher informational content of the higher organisms will most assuredly be measured as greater randomness.

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Secondly, the failure to find a completely suitable stochastic model for the bacteria and archaea does not mean that randomness does not operate on their chromosomal arrangement. What it means is that there are functional limitations caused by the differences in how bacteria and archaea, on the one hand, and eukaryotes, on the other, process the DNA's information into protein. Those differences cause bacteria to appear less random, but this also means that they appear more ordered. And since order is a property of static things (like highly ordered, but totally inert, mineral crystals), more order in a living system is an indication of less complexity.

Methods for determining design need to be robust enough to conclude that the structure of a cell is designed. If it cannot, then one of two propositions therefore must be true: (1) either the cell is not designed; or (2) the methodology is flawed. We prefer to believe that the current Dembski methodology is flawed.

The preference for short gene strings, which our data demonstrates, may have implications for origin of life issues. It shows that life at this level of structure is built up of repetitive, simpler systems which are amalgamated into the larger unit. Contrary to the perception that higher organisms should use larger and more complex organizational systems for their genomic information, they actually use a system of gene strings which are small and simple rather than large and complex.

Finally, Christian apologists need to incorporate chance and randomness into their world views. It is clear that the Bible teaches that God controlled chance and randomness at several crucial junctures in history. If he did this, then the controlling of chance and randomness in biology should be equally possible. Apologists should not ignore the observational fact that as we go from simple creatures to the more complex organisms, chromosomal organization appears to be much more influenced by random processes. Indeed, chromosomal structure at the complex end

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of the spectrum matches the predictions of very simple stochastic models. It does no good to claim that chance or random processes cannot produce more complex organisms, when those very organisms are measurably more random than are the simpler creatures.

In conclusion, we should all remember what God said to Job: "'Will the one who contends with the Almighty correct him? Let him who accuses God answer him!' Then Job answered the LORD: 'I am unworthy—how can I reply to you? I put my hand over my mouth. I spoke once, but I have no answer—twice, but I will say no more'" (40:2, NIV).

It seems to us that God is not scolding Job for his ignorance but rather for his lack of humility—by attempting to explain how God conducts his activities. Applied to our present discussion, we would dare to suggest that a significant degree of humility is needed by all Christians, to accept—in agreement with various biblical teachings—that God is perfectly capable of fully designing the various life forms we observe, however he pleases, in a way we surely do not understand, while presenting his handiwork to us mere mortals in a format that shows strong evidence of randomness. ❁

Notes

[1]Henry M. Morris, "The Compromise Road," *Impact* 177 (March 1988): I, ii.

[2]Lee Spetner, *Not by Chance* (Brooklyn: The Judaica Press, Inc., 1998), vii.

[3]Jonathan Wells, "Making Sense of Biology," *Touchstone* (July/August 1999): 55.

[4]Adnan Oktar, "The Evolution Deceit," www.hyahya.org/16understanding03.php, accessed Sept. 23, 2001.

[5]William A. Dembski, "Signs of Intelligence," *Touchstone* (July/August 1999): 80.

[6]——, "On the Very Possibility of Intelligent Design," in J. P. Moreland, ed., *The Creation Hypothesis* (Downer's Grove: InterVarsity Press, 1994), 116; A. E. Wilder-Smith, *The Natural Sciences Know Nothing of Evolution* (San Diego: Master Books, 1981), 85; Phillip Johnson, "Creator or Blind Watchmaker," *First Things* (Jan. 1993): 12; Phillip Johnson, "Immodest Ambitions," *Books & Culture* (Sept. Oct. 1995): 29; Michael Behe, *Darwin's Black Box* (New York: The Free Press, 1996), 191–2; Robert F. DeHaan and John L. Wiester, "The Cambrian Explosion," *Touchstone* (July/August 1999): 65.

[7]See Exod. 28:30; Lev. 8:8; Deut. 33:8; Ezra 2:63; and Neh. 7:65. See also "Urim and Thummim" Trent C. Butler, ed. *Holman Bible Dictionary* (Nashville: Holman Bible Publishers, 1991) for more on this.

[8]Dr. Robert Gange, *Origins and Destiny* (Waco, TX: Word, 1986), 73.

[9]Available in the English translation of *Foundations of Probability Theory* (New York: 1950).

[10]Gregor Mendel *Experiments in Plant Hybridization*, (1865).

[11]www.ncbi.nlm.nih.gov/PMGifs/Genomes/allorg.html

[12]Most techniques used are found in any statistical textbook. Some special techniques of note are: F. N. David, "A Note on the Evaluation of the Multivariate Normal Integral," *Biometrika* 40 (1958): 458–9; W. F. Sheppard, "On the Application of the Theory of Errors to Cases of Normal Distribution and Normal Correlation," *Philosophical Transactions of the Royal Society of London Series A* 192 (1899): 101–67; W. F. Sheppard, "On the Calculation of the Double Integral Expressing Normal Correlation," *Transactions of the Cambridge Philosophical Society* 19 (1900): 23–66.

[13]Clyde Hutchison, Michael Montague, Glenn Morton, Michelle Opp, Gabor Pataki, and Gordon Simons (2001, unpublished manuscript); see also Gordon Simons and Glenn Morton, "The Gene-Orientation Structure of Eukaryotes," *Journal of Theoretical Biology* 22, no. 4 (2003): 471–5.

[14]Henry M. Morris, "The Power of Energy," in Walter E. Lammerts, ed., *Scientific Studies in Special Creation* (Grand Rapids: Baker Book House, 1971), 66; Randy L. Wysong, *The Creation-Evolution Controversy* (Midland, MI: Inquiry Press, 1976), 257; Henry Morris, *The Remarkable Birth of Planet Earth*, (Bloomington, MN: Bethany House, 1972), 1; ——, *Creation and the Modern Christian* (El Cajon, CA: Master Book Publishers, 1985), 214–5; A. E. Wilder-Smith, *Man's Origin, Man's Destiny* (Wheaton, IL: Harold Shaw, 1968), 57.

[15]Lee Spetner, *Not by Chance* (Brooklyn: The Judaica Press, Inc., 1998), vii.

[16]Wells, "Making Sense of Biology," 55; see also Rick Wade, "Defeating Darwinism," in Ray Bohlin, ed., *Creation, Evolution, & Modern Science* (Grand Rapids, MI: Kregel Publications, 2000), 98.

[17]Percival Davis and Dean H. Kenyon, *Of Pandas and People* (Dallas: Haughton Publishing Co., 1993), 66.

[18]Hubert Yockey, *Information Theory and Molecular Biology* (Cambridge: Cambridge University Press, 1992), 81–2.

[19]Dembski, "Signs of Intelligence," 79.