Book Review

Not by Chance! Shattering the Modern Theory of Evolution

(Brooklyn, NY: The Judaica Press, 1997) by Lee Spetner, Ph.D. 272 pages, $14.95

Reviewed by Ashby L. Camp, J.D., M.Div.

Lee Spetner received the Ph.D. degree in physics from MIT in 1950 and joined the Applied Physics Laboratory at Johns Hopkins University the following year. He spent most of his career doing research and development on information processing in electronic systems, and in teaching information and communication theory. In 1962 he accepted a year's fellowship in the Department of Biophysics at Johns Hopkins where he was to solve problems in the extraction of signal from noise in DNA electronmicrographs. During that fellowship, he learned much about biology.

Between 1964 and 1970, Dr. Spetner published several papers in the professional literature dealing with various aspects of evolutionary theory. His work appeared *in Journal of Theoretical Biology, Proceedings 2nd International Congress an Biophysics, IEEE Transactions on Information Theory,* and *Nature.* He then returned to his regular work, but he continued to follow the developments in molecular biology and genetics. His vast reading on evolution has gained him a true command of the subject.

The first three chapters of the book are introductory but important. Chapter 1 provides some historical background, chapter 2 explains some essential facts about biology, and chapter 3 describes the neo-Darwinian theory of evolution.

The core of Dr. Lee Spetner's challenge to neo-Darwinian theory (NDT) is in chapters 4 and 5. He points out that evolutionists have repeatedly stressed that NDT is based on random genetic changes, meaning changes which are not related to the needs of the organism, and not biased toward adapting the organism to its environment. The question is not whether evolution is random but whether the genetic variation on which natural selection works is random. When evolutionists say that evolution is not random, they mean only that natural selection produces a non-random result from the random genetic variation.

The second requirement of NDT is that the random mutations which fuel it must also, on average, add information to the genome. If evolution built up the complexity of life, then it must also have built up the information underlying that complexity.

A minority of evolutionists say that macroevolutionary change is more often a single, large, random change than it is a chain of small ones. They say that large changes in phenotype come mainly from mutations of regulatory genes, but Spetner rejects the notion that such genetic rearrangements can serve as the random variation required by NDT. He does so for two reasons:

> (1) There is good reason to believe that these complex genetic rearrangements are not random. They seem to be deliberate acts performed on behalf of the cell (or the organism) which involve special enzymes and structures. Insertions are made so they can be precisely removed, and inversions are made so they can be precisely reversed.
>
> (2) The claim that evolution is due to such mutations of regulatory genes does not account for how information can build up in the genome. The only kind of regulatory changes evolutionists have suggested so far, insertions and inversions, are ways of turning ***existing*** genes OFF and ON. If they turn ON a regulatory gene, they can bring into play a complex function or a whole system of functions, but the information must already be in the genome.

Most evolutionists, on the other hand, hold that a large evolutionary change occurs through a long chain of small steps (cumulative selection). They maintain that the mutations in these small steps are copying errors, which everyone agrees are random. But, according to Spetner, the chance of getting the necessary mutations is just too small if it's done through cumulative selection. (See sidebar on page 5 for Spetner's probabilistic analysis.)

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—Spetner, 1997

Regarding the requirement that the random mutations which fuel NDT must, on average, add information to the genome, Spetner examines the examples of adaptive mutations that are touted as prototypes of macroevolution (resistance of bacteria to antibiotics, resistance of insects to pesticides, breeding of "quantitative traits," and adaptation of soil bacteria to new nutrients) and finds them all wanting. He explains how none of these mutations adds new information or any new molecular capability. Instead, they all destroy information. In a memorable line, he says, "Whoever thinks macroevolution can be made by such mutations is like the merchant who lost a little money on every sale but thought he could make it up on volume."

Spetner points out there are several examples of mutations that permit bacteria to live on a new nutrient and that seem to add a lot of information. In fact, some experiments have shown the introduction of an entirely new enzyme (as opposed to the degradation of an existing one), and

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Spetner's Probabilistic Analysis

Spetner presents the following analysis showing why the probability of getting the needed mutations through cumulative selection is just too small.

G. Ledyard Stebbins, one of the architects of NDT, has estimated that to evolve a new species would require about 500 steps. For each of these steps, a mutant with a positive selective value must appear and must be lucky enough to survive and to eventually take over the population. Since Stebbins is an expert in the field, and Spetner knows of no prominent evolutionist who disagrees with his estimate, he accents this figure as a reasonable ***typical*** value.

Assuming that for each step there is only ONE point mutation (one specific base of one specific nucleotide) that has a positive selective value, the odds that this specific nucleotide base will randomly appear depends on how may reproductions occur during each step. Based on George Gaylord Simpson's estimates regarding the allegedly well understood evolution of the horse, it can be calculated that there are about 50 million births during each evolutionary step.

The chance that a specific nucleotide will mutate in one birth is known to be 1 in 10 to the 10th power (Spetner did some of the original work on this). So the chance this will occur over 50 million births (50,000,000 X 10 to the minus 10th power) is 1 in 200. But since there is a roughly equal chance that the base of the nucleotide will change to any one of the other three bases (the four nucleotide bases are adenine, guanine, thymine, and cytosine), the odds of getting a specific change of a specific nucleotide is l/200 X l/3 = l/600 *(i.e.,* one chance in 600).

Since such a mutation (a point mutation) is the smallest possible mutation, any selective value it has must also be small. Simpson said a "frequent [selective] value" of evolutionary mutations is about 0.1%, meaning the mutant's average number of surviving offspring is 0.1% higher than the rest of the population. Sir Ronald Fisher, one of the world's experts on the mathematics of evolution, showed the odds that a single mutation having a selective value of 0.1% will survive are 500 to 1 against its survival (because most mutants, like most other members of a population, are wiped out by random effects).

So the chance that a specific change (base) of a specific nucleotide will both occur during a step (1/600) and survive to take over the population (1/500) is 1/300,000. For this to happen 500 times in a row, the number of steps estimated to be necessary to achieve a new species, the odds are 1/300,000 multiplied by itself 500 times. The odds against that happening are about 3.6 X 10 to the 2738th power to one, or the chance of its happening is about 2.7 X 10 to the minus 2739th power. This is, of course, an essential impossibility.

The evolutionist naturally counters that it is unreasonable to assume there is only ONE point mutation at each step that will have a positive selective value. If at each of the steps in the process there is more than ONE potential adaptive mutation, then the odds for evolution improve accordingly. So Spetner investigates how many possible adaptive mutations there must be at each step for evolution of a species to have a "reasonable" chance.

For evolution to have a one in a million chance (1 X 10 to the minus 6th power) of producing a new species through 500 steps, the odds that a specific change of a specific nucleotide will both occur during a step and survive to take over the population must be 0.9727 for each step, or about 36 out of 37 (the probability of each step must be multiplied by itself 500 times to reach the probability of all 500 steps occurring). For that to be true, for each step there must be 1,080,000 potential copying errors with a positive selective value. In other words, only with that many possible adaptive mutations does one reach the necessary probability (0.9727 or 36 out of 37) that at least one of the potential adaptive mutations will both occur and survive to take over the population.

But NDT can find no refuge here. If at each of the 500 steps in the transition from one species to the next there are over one million potential adaptive mutations, it would be essentially impossible for the same trait to ever evolve in two different species. The amount of freedom is just too great. Yet, evolutionists claim this has happened repeatedly in what is called convergent or parallel evolution. There are thousands of examples, but some of the more striking involve ultrasonic echolocation systems, electrostatic imaging systems, electric generators, visual systems, and the mammalian brain.

For convergent / parallel evolution to occur, two lines of descent would have to make the same random choices at many of the 500 steps of speciation. With one million potential choices at each step, if only 100 of the 500 choices needed to be the same, the odds against it would be one in 10 to the 600th power. And this is only for convergence in a single species transition. For convergence of a complicated organ such as a wing or a kidney or an eye, the probability would be much smaller because one would need to allow for many species and thus many thousands of steps.

Evolutionists object to these calculations on the basis that many genotypes may lead to the same phenotype, so the number of choices at each step (a million) is too high. But since genotype determines phenotype, freedom in genotype choices must translate to some degree into freedom in phenotype choices. If the one million genotype choices at each of the 500 steps of transition to a new species equate to only 10,000 phenotype choices, the total number of branches in the 500 steps is still 10 to the 2000th power. The odds against coming out at the same place twice would still be essentially impossible.

Moreover, current research is showing that phenotype convergence does imply genotype convergence. The genes controlling eye development in both insects and vertebrates have been identified, and they are 94% identical. This makes convergence so improbable that the authors of the study say the consensus view that the vertebrate eye and the insect eye evolved independently "has to be reconsidered." Another recently found example of convergence in the genotype involves the enzyme lysozyme in the cow and langur monkey (to which Spetner devotes several pages).

The diversity that exists in potential recombinations of existing base sequences does not, as is sometimes claimed, avoid the probability problem. Given that about 6,700 of the approximately 100,000 genes of the human genome come in two versions in the same person (one of each member of a chromosome pair), there are 10 to the 2017th power different possible combinations of genes. In order for one of these recombinations to yield a base sequence with a positive selective value through the relatively tiny number of recombinations that can actually be achieved in an evolutionary step, a great many of the potential recombinations (10 to the 1998th power, ***without*** considering the survival factor!) would have to be adaptive. If that is the case, there is again no place for convergent evolution.

— Ashby L. Camp

Questions from the Internet

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of an organ's function (and ultimately disappearance of the organ itself) certainly doesn't fit this definition.

How do evolutionists know that the appendix is not, in fact, a nascent organ; ***i.e.,*** one that is just beginning to develop in humans? Or, perhaps monkeys, which have no appendix, are more evolutionarily advanced than lemurs, apes, and humans.

Your statement that the human appendix is useless shows that you have been duped into **believing in** evolution. At one time, it has been said, as many as 180 organs were claimed by evolutionists to be vestigial. Medical research has shown that virtually all of these have one or more function. The evidence thus seems to suggest that there are no truly functionless organs, only those whose function we have not yet discovered. There are also organs which are essential at a certain stage of development, but may have no apparent function later in life.

Since you are "open-minded," would you consider your belief in evolution to be falsified if a function for the appendix can be demonstrated? Conversely, would you consider creation to be confirmed if functions are found for organs previously declared to be vestigial by evolutionists? I doubt it, because your religion (that in which you place your faith) is atheistic evolution.

One problem experienced by evolutionists, such as yourself, is the mistaken notion that the appendix once had the function of the cecum (digestion of cellulose). However, man and many herbivorous mammals have both a cecum AND an appendix. That the appendix does, indeed, have a function was noted by the prominent evolutionary paleontologist Romer (1970, p. 344). Speaking of the cecum, he said:

> In man it [the cecum] terminates in the narrow vermiform appendix. This is frequently cited as a vestigial organ supposedly proving something or other about evolution. THIS IS NOT THE CASE; a terminal appendix is a fairly common feature in the cecum of mammals... *(emphasis added)*

Many sources (encyclopedias, textbooks, etc.) still erroneously state that the appendix is useless. Interestingly, the ***Grolier Multimedia Encyclopedia*** states in one place that "In humans the cecum and appendix have no important function," and in another place that "the appendix is now thought to be one of the sites where immune responses are initiated" (Hartenstein, 1995). The appendix is, in fact, loaded with lymphatic tissue, making it a part of the body's vast immunological system.

For additional information about the function of the appendix, and for the most thorough treatment of the entire subject of vestigial organs, the reader is referred to the CRS monograph by Bergman and Howe (1990).

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recent experiments have shown that bacteria can mutate to produce new functions when they are needed. BUT these mutations were not point mutations. Rather, they appear to be mutations which are triggered by environmental stimuli and which turn ON ***existing*** genes. In other words, they are not random and do not add information. He believes this kind of plasticity may be ***designed*** into living creatures to allow them to exploit changing environments, a subject he explores in chapter 7 under the title "the nonrandom evolutionary hypothesis."

Chapter 6 is devoted to exposing the flaws in Richard Dawkins' popular ode to naturalism, The ***Blind Watchmaker.*** Spetner ends that chapter with another great line. He says, "The dust jacket of Dawkins' book says, 'There may be good reasons for belief in G_d, but the argument from design is not one of them.' I would put it differently: There may be good reasons for being an atheist, but the neo-Darwinian theory of evolution is not one of them."

Chapter 8 is an epilogue in which Spetner discusses the implications of the conclusion that evolution cannot be random. Unlike NDT, which denies creation, he says his nonrandom evolutionary hypothesis poses no contradiction to creation. Rather, it is perfectly consistent with the suggestion of Rabbi David Luria (in his commentary to the Midrash) that 365 basic species of beasts and the same number of birds were originally created; all the others derived from these.

The book includes a 32-page appendix which explains in more detail some of the biological functions discussed in the book. It ends with an 18-page section of references and a 10-page index of people and subjects.

I consider this book to be ***must reading*** for those interested in the creation-evolution issue.

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