The Evolution Revolution: Why Thinking People are Rethinking the Theory of Evolution

by Lee Spetner

reviewed by David E Levin

The Evolution Revolution is an abbreviated reprise of Lee Spetner's Not by Chance! (1997), in which he introduced his concept of the “nonrandom evolutionary hypothesis” (NREH). Spetner is a retired PhD physicist and Jewish creationist, who argued in his first book that the mechanism central to the modern evolutionary synthesis—random mutations coupled with natural selection—is incapable of adding new information to the genome. He asserts that the evolutionary appearance of new biological features must involve alterations to the genome that were pre-programmed, presumably by the Creator. Spetner proposes that such alterations are induced by environmental stresses and result in adaptation to those stresses. This is essentially the application of the Lamarckian idea of inheritance of acquired characteristics to alterations of the genome. Spetner's mechanism works like this: identical, environmentally induced genomic alterations are triggered in a large fraction of a population when the population is exposed to the stress condition. This, the author claims, could “account for much of the rapid evolution that has been observed” (page 43) by scientists.

In this new book, Spetner offers a poorly-written rehash of his idea that organisms respond to environmental signals by rearranging their genomes in a pre-programmed and adaptive way. The addition this time around is his claim that a variety of scientific discoveries made in recent years has generated evidence in support of his hypothesis. However, no such evidence exists.

He makes much of the fact that bacteria stressed by starvation or other growth-inhibitory conditions can activate a “hyper-mutation” system that elevates the rates of both point mutations and larger genomic rearrangements, such as duplications and deletions, as well as the activation of mobile DNA elements. This process can result in adaptive alterations that enable rare individuals with the appropriate genetic alterations to survive and reproduce under conditions in which the rest of the population cannot. Spetner would have the reader believe that this process is in some way directed, or targeted. It is not. Rather, elevated rates of mutations and rearrangements are detected across the genome, not just at sites that might result in an adaptive change.

Throughout the book, Spetner rejects common descent and asserts incorrectly that we can only learn about evolution from the study of evolutionary events that are rapid enough for us to observe, offering an entire chapter entitled “Rapid evolution is happening today.” A reader might reasonably ask why, if we can observe many examples of what Spetner de-
scribes as rapid evolution, does he reject common descent? Here, he deploys the common creationist trope of accepting “microevolution,” but not “macroevolution,” with the added twist that in his view, microevolution is largely the result of a built-in ability of organisms to alter their genomes to respond adaptively to environmental stimuli. Such adaptations would necessarily, by Spetner’s thinking, be limited in terms of their diversity. Thus, observable evolution fits well with Spetner’s talmudic view, adopted from a nineteenth-century rabbi, that “[t]here were 365 basic families of animals created, and the same number of bird families. All others evolved from these, but not through random mutation and natural selection. Rather, they evolved by undergoing natural changes induced by their environment” (page 109).

Spetner’s real problem becomes clear when he tries to apply this thinking to plants and animals, where, as is the case in any sexually reproducing multicellular organism, heritable alterations must reach the germline to affect the next generation. He posits that environmental stresses induce hormones, which somehow induce adaptive genetic rearrangements within the sperm or eggs of the stressed individual, so that the adaptation can be passed on to the next generation. This suggestion, for which there is no evidence, strains the bounds of credulity.

Spetner offers numerous examples of “rapid evolution.” Some of these examples, he acknowledges, might be explicable in terms of natural selection acting on pre-existing genetic variation, but he offers other examples failing to recognize that they do not involve heritable changes at all. For instance, he provides what he describes as “a good example of NREH in action” (page 64), in an endangered species of pupfish that lives in the inhospitable environment called Devil’s Hole, near Death Valley. This geothermal pool has little by way of food, and as a guard against extinction, scientists have relocated some individuals from this population in recent years to artificial refuges with similar, but not identical, environments. It was soon observed, however, that pupfish within these refuges had undergone rapid and significant morphological changes compared to the original population. Spetner suggests that the morphological shift is the result of environmentally triggered, heritable genetic alterations. But in fact, follow-up laboratory experiments, conducted by biologist Sean Lema, have revealed that this phenomenon is nothing more than an environmentally induced change in morphological development, which can be manipulated in fry from a single genetic stock exposed to environments with varied temperature and food rations—in other words, no genetic mutations were involved, rather, developmental gene expression was affected by the environment. Spetner asserts disingenuously that “the DNA of those in the refuges differed from those in Devil’s Hole” (page 65), whereas the actual finding was that their genomes were nearly identical. Contrary to Spetner’s assertion, this is a case of developmental plasticity, not an example of rapid evolution. The fact that Spetner highlights this as a key example of his NREH demonstrates how completely bereft of evidential support his idea remains. It is clear that Spetner, who has no training or expertise in biology, has combed through the biological literature in search of any interesting or anomalous report that he imagines might support his idea that adaptive genomic alterations have been pre-programmed into the genomes of all species.

In his quest to discredit modern evolutionary theory, Spetner repeatedly insists that “the scientific literature records no example of even one random mutation that adds heritable information to the genome” (page 110), an astonishing claim by any measure. He is able to
make this assertion by couching the concept of added information in terms of increased molecular “specificity”, which he repeatedly redefines to suit the example at hand. In one example, he describes an evolutionary series of mutations in a soil bacterium that enabled it to grow on a medium of the sugar xylitol, a substance that the bacteria cannot normally use as a carbon source. One of the mutations was in an enzyme, called ribitol dehydrogenase (RDH), which is involved in the degradation of ribitol, a similar sugar that the organism can use. The mutant form of RDH has an increased ability to act on xylitol and a decreased ability to act on ribitol. Spetner asserts here, based on the enzyme's ability to use a third sugar as substrate, that this mutant enzyme has a “reduced specificity” relative to the starting enzyme and that a loss of specificity (defined in this way) amounts to a loss of information. However, he later changes his definition of specificity when discussing the evolution of antibiotic resistance. He explains that the antibiotic streptomycin interferes with bacterial protein synthesis by binding at a specific site on ribosomes and causing them to increase the error rate in the synthesis of proteins (that is, misincorporation of amino acids). Mutations in a particular ribosomal subunit confer resistance to streptomycin by blocking binding of the drug to the ribosome. Spetner asserts here that the mutation that confers streptomycin resistance ruins the streptomycin binding site, thereby “destroying the specificity of the protein” (page 119). But he has arbitrarily changed his definition of specificity to suit his need. Based on his definition from the earlier example, the streptomycin-resistant ribosome has increased specificity, because it binds one fewer molecules than the starting ribosome. By dishonestly shifting his definition of specificity at every turn, Spetner is able to dismiss any example of molecular evolution one might provide.

Finally, a reader would have thought that any serious critique of the science that underwrites the conclusion of common descent would have addressed the evidential cornerstone of that conclusion—the twin nested hierarchies observed in the comparisons of both morphological and genomic features. But Spetner failed to address this issue at all. He does not seem to understand that trained anatomists have organized all living things into nested groups based upon shared ancestral features and that the existence of such nested groups is a strong prediction of common descent. He further fails to acknowledge that recent advances in molecular technologies have allowed the comparisons of entire genomes and that those comparisons also generate nested hierarchies of genomic differences between species. Rounding out his apparent lack of expertise on this front, Spetner fails to recognize that the nested hierarchies produced by anatomical and genomic comparisons agree with each other astonishingly well. How can anyone pretend to set out a critique of common descent without addressing this central issue?

Instead, Spetner takes the approach of attacking the phylogenetic tree by presenting a deeply flawed understanding of convergent evolution. He claims that the notion of convergence was “invented solely to avoid addressing the failure of the phylogenetic tree to support Common Descent” (page 89). By confusing the concepts of analogous and homologous structures, he manages to make comical lateral connections between highly divergent phylogenetic groups, asserting on their basis that there is no unique phylogenetic tree. In perhaps the most amusing of his examples, Spetner compares the mammalian ear to the hearing system of katydids, a group of insects related to crickets. In mammals, the tympanal membrane (the eardrum) is connected to the frequency-detecting organ of the inner ear (the cochlea) by a lever system of three small bones in the middle ear. Katydids
possess a tympanal membrane functionally analogous to the eardrum; a tympanal plate, which is analogous to the middle ear bones; and a fluid-filled acoustic vesicle, which is analogous to the cochlea. The broad similarities in these acoustic systems are regarded as an example of convergent evolution because insects and mammals have converged upon a similar solution to the problem of sound detection through the use of unrelated structures. For example, insects do not have bones and their tympanal plates are made of the same material as their hard outer cuticle. Most remarkably, Spetner neglects to mention that the insect “ears” are located in their front legs. It is nothing short of astonishing that Spetner appears not to grasp the fundamental concept that descent with modification produces homologous structures, not analogous structures.

It is clear from this book that Spetner does not take a scientific approach to his scholarship. His presented arguments against common descent and evolutionary mechanisms are at once both deceptive and ignorant. He has placed his religious beliefs above his critical analysis of data, selecting only the bits of information that he can distort to fit his ideas. Invariably, close examination of the primary literature relating to his cited examples of NREH reveals either mechanistic ambiguity or outright contradiction of his hypothesis. Most non-scientists, however, will find his abbreviated technical discussions inaccessible and will not be motivated to read more deeply. Thus *The Evolution Revolution*, like most books of its kind, has the capacity to reinforce the religious beliefs of those who do not wish to understand science, but does not have the potential to influence the thinking of scientists.

**REFERENCES**


**ABOUT THE AUTHOR**

David E Levin is Professor in and Chair of the Department of Molecular and Cell Biology at Boston University’s Henry M Goldman School of Dental Medicine.

**AUTHOR’S ADDRESS**

David E Levin  
Department of Molecular and Cell Biology  
Boston University Henry M Goldman School of Dental Medicine  
72 E Concord St, Evans 4th floor  
Boston MA 02118  
delevin@bu.edu