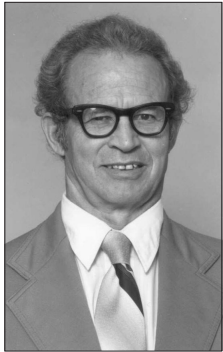


In Defense of Intelligent Design



I believe my many years of training and experience [in biochemistry] qualify me to evaluate the scientific evidence [used in the arguments for and against design].

Authors of two recent books, Kenneth Müller (*Finding Darwin's God*)¹ and Michael Ruse (*Can a Darwinian be a Christian?*)² have been very critical of Intelligent Design, and especially of Michael Behe's arguments for design in *Darwin's Black Box*.³ Many of their criticisms dealt with biochemical aspects of design, where I believe my many years of training and experience qualify me to evaluate the scientific evidence.

Ancestral descent and evolution. One of the key components of the Darwinian Theory of Evolution has been the concept of ancestral descent. This concept holds that in the course of evolution, there has been a gradual change from primitive organisms, such as bacteria, to more advanced organisms, such as mammals, and that these changes can be explained by chance events (mutations, gene duplications, etc.).⁴ The changes would then be fixed in organisms by natural selection. The ancestral descent concept holds that these changes would be demonstrable both in the structures (morphology) of organisms and in the cellular biochemistry used for fundamental processes, such as the trapping of energy. Ruse and Miller believe that if these latter changes can be accounted for by chance events, they will have struck a severe blow to Behe's principle of irreducible complexity, which he uses to argue for Intelligent Design.

Tricarboxylic acid cycle and a proton pump. First let me review the metabolic systems that Miller and Ruse have chosen in their attempt to validate their thesis regarding ancestral descent. In the 1930s, Hans Krebs discovered, through a series of carefully designed experi-

ments, what is now known as the Krebs tricarboxylic acid cycle. This cycle has been shown subsequently to be the most important pathway in vertebrates for converting the potential energy of a number of different nutrient compounds into the readily available energy of adenosine triphosphate (ATP). The mechanism of coupling the oxidative sequences in this pathway to formation of ATP is known as a respiratory chain and involves enzymes known as cytochromes, various cofactors, and a proton pump. Even though the respiratory chain often is considered separately from the tricarboxylic acid cycle in textbooks, within the mitochondria of cells, they are very closely linked in their function. These are indeed two very important systems in vertebrate metabolism. Did these systems and their component enzymes originate in bacteria and evolve by chance into the more complex forms found in mammalian cells? Let us examine what the evidence shows and see if the mammalian system has been shown to have been cobbled together by chance from some bacterial precursor enzymes.

Their Evidence for Ancestral Descent

Tricarboxylic acid cycle. Ruse and Miller both cite a 1996 paper by Melendez-Hevia, et al. on the tricarboxylic acid cycle enzymes,⁵ and Miller cites a 1998 paper on the proton pump by Musser and Chan in support of the ancestral descent thesis.⁶ In regard to the first of these papers, Ruse notes:

Yet the cycle did not come out of nowhere. It was cobbled together out of other cellular processes which do other things ... Each one of the bits and pieces of this cycle exists for other purposes and has been co-opted for the new end (p. 115).

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Miller is equally enthusiastic in his claim that this research paper supports the hypothesis of ancestral descent. He says:

The Krebs cycle is a complex biochemical pathway that requires the interlocking, coordinated presence of at least nine enzymes and three cofactors. And a Darwinian explanation for its origin has now been crafted (p. 151).

In this communication, I wish to show that Ruse and Miller are dealing with this topic at a superficial level. When one examines the data closely, their arguments are not adequate to explain their hypothesis. First let me note several types of studies that are essential to Miller's claim of a good "Darwinian explanation" for ancestral descent of the enzymes of the tricarboxylic acid cycle. They are:

1. There would of necessity be strong sequence similarity of each of the enzymes in the cycle and the claimed ancestral enzymes found in bacteria, where the enzymes were functional for other purposes.
2. There would need to be a proposal of some kind of phylogenetic tree showing plausible steps from apparent bacterial ancestral enzymes to the enzymes of a functional Krebs cycle. This would need to be supported by amino acid or DNA similarities.
3. Since Krebs cycle enzymes, when functioning in oxidative metabolism, are found in mitochondria of eukaryotic cells (cells with a nucleus), there would need to be a mechanism for incorporation of the enzymes into the correct structural relationships in the mitochondrial matrix.
4. Since bacterial genes are predominantly found as circular DNA, there would need to be an incorporation of these genes into the chromosomal DNA of the cell nucleus. In a few instances, the ancestral bacterial genes would need to be transferred to the circular DNA of the mitochondria, rather than the cell nucleus.

Melendez-Hevia, et al. do not deal with any of the questions that I have posed, although they do indicate in their second and third stages (p. 294), the necessity of organization for all of the components of the cycle. Organization and regulation are absolutely essential for a functioning Krebs tricarboxylic cycle. The organization and regulatory stage would, of necessity, include incorporation of the various heme enzymes and cofactors that are essential to the trapping of cellular metabolic energy as adenosine triphosphate. With thousands of different species of bacteria for consideration, only one, an anaerobic bacterium, *Desulfotomaculum*, is listed by Melendez-Hevia, et al. by name. A reasonable proposal would surely list possible ancestral bacteria showing that all of their necessary enzymes were gradually incorporated into cells of a single ancestral species.

The major thrust of these authors in this paper is on the types of compounds and the types of reactions involved in

the tricarboxylic acid cycle and why the particular reactions are the most appropriate. The authors do not deal with whether these compounds were selected by chance or chosen by a designer. They do make some interesting thermodynamic and kinetic observations about why certain alternative pathways would not have evolved (or have been designed). Their proposed stages (p. 294) in the history of life are interesting, but are unproven. Their proposed "Rules for Designing Metabolic Pathways" (p. 297) are reasonable for any pathway dependent on an evolutionary sequence, but, in most cases, also would apply for a sequence brought about by Intelligent Design.

A philosophical question. A most important philosophical question seems to have been overlooked by these authors. Providing an apparently feasible explanation about how something may have happened does not prove that it did happen that way! Sometimes, explanations that appear feasible on the surface can be shown to be quite inadequate when one digs more deeply. Since Melendez-Hevia, et al. did not deal with any of the types of studies that I have suggested, I must consider their studies as failing to provide the "proofs" that are claimed for them by Ruse and Miller.

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Another point needs to be made clear for anyone not familiar with the metabolic details of biochemistry. Interrelationships of different metabolic pathways in biochemistry are common. Amino acid metabolism is closely linked to carbohydrate metabolism and has been taught that way for fifty years or more, so the linking of the metabolism of amino acids to Krebs cycle enzymes is not new. Oftentimes the same enzyme or enzyme complex may be used in linking pathways. Consequently, indications of interrelationships of Krebs cycle enzymes with enzymes of amino acid metabolism is not necessarily an argument that the latter have an ancestral relationship to the Krebs cycle enzymes.

Proton pump. Kenneth Miller (*Finding Darwin's God*, pp. 149–50) gives a second illustration of evolution utilizing existing components in the formation of a proton pump. This pump is an important component of a respiratory chain in vertebrate cells. Miller notes the work in 1998 of Musser and Chan, who were able to produce in impressive

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detail, "an evolutionary tree constructed using the notion that respiratory complexity and efficiency progressively increased throughout the evolutionary process."⁷

The paper of Musser and Chan is a very careful study of the different enzyme complexes utilized for proton translocation in bacteria as well as in cells of mammals, plants, and fungi. They note the differences in hydrogen donors in these different organisms as well as the difference in the terminal proton acceptor. They also note the similarities in the heme protein catalytic group used in all of these organisms. With an evolutionary tree based on ribosomal RNA sequences as a model, the authors propose a similar tree for the evolutionary development of the components of the proton pump. However, the authors note that they use only minimal amino acid sequence information of the heme proteins in their analysis. Therefore, it should be emphasized that their phylogenetic tree is based on ribosomal RNA sequences rather than sequences in the actual enzymes of the proton pump.

Despite the differences in the bacterial proton pump and the proton pump found in mammals, the authors do note a number of developmental similarities that suggest an ancestral relationship. However, they assume that any such relationship could come about only by chance events (gene duplications, mutations, etc.). They do not eliminate the possibility of design because they do not consider it. In my view, all of the similarities they note may very readily be explained as due to a common creator (designer). Until protein and gene sequences are more carefully examined, it is premature to claim, as does Miller, that the question of the origin of the vertebrate pump has been resolved. Musser and Chan do discuss the importance of the spatial relationships of the various components in the mammalian mitochondrial pump. However, they do not suggest how this spatial organization might have developed from the structures of the different components as they are found in bacteria.

Conclusion

In summary, Musser and Chan have suggested a possible ancestral relationship of a bacterial proton pump to the mammalian proton pump in the mitochondrial respira-

tory chain. However, they have not presented protein or DNA sequence studies that would be essential for placing their hypothesis of an ancestral relationship on a firmer basis. If the evolutionary model of formation is correct, the authors would need to demonstrate verifiable evolutionary pathways utilizing only chance events. It also should be emphasized that similarities do not prove that relationships have come about by chance. Similarities may also be a consequence of a common creator or designer.

Comments regarding Intelligent Design.

After dealing with some of the criticisms of Intelligent Design, I believe a few additional positive comments are warranted. Many writers assume that a Creator would use only fiat creation, i.e., creating entire organisms. However, there is no reason to limit the creative activity of a Creator to fiat creation. In some cases, the jumps necessary to bridge gaps in phylogenetic relationships might be brought about by relatively small changes in chromosomal DNA, particularly with changes in developmental genes. Unless one can make probability estimates for the possibility of these changes, it may be nearly impossible to know which changes were a consequence of chance mutations and which were due to modifications by a designer. My view, which I now refer to as a Design Theory of Progressive Creation, never postulates that all changes must be due to specific acts of design,⁸ whereas the traditional evolutionary view insists that all changes must be a consequence of chance (usually gene duplication, mutation, and natural selection).

The hypothesis that enzymes or other protein molecules might be built up from smaller modular units at the level of genes is worthy of serious consideration. I have considered possible movement of modular units in several of my papers.⁹ However, if modular units are used, one still must postulate some source of information in nonmodular portions of protein molecules, and controls for regulating movement of modules around in cells of higher organisms are very stringent. The overall probability of putting a protein together by combining modular units, each of which had been formed separately, is no different than putting a protein together one amino acid at a time. All studies carried out so far indicate the extremely low probability of obtaining a single protein

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by chance. For cytochrome *c*, a small protein with about one hundred amino acids, the probability of starting with only L-amino acids is 2×10^{-65} .¹⁰ Present estimates for the human genome indicate that 30,000 to 40,000 genes are present. Since the cytochrome *c* gene is smaller than average in size, and all evidence indicates that production of other genes for proteins would have corresponding low probabilities for chance formation, how can one not postulate a designer? ☆

Notes

- ¹Kenneth Miller, *Finding Darwin's God* (New York: Harper-Collins Publishers, 1999).
²M. Ruse, *Can a Darwinian be a Christian?* (Cambridge: Cambridge University Press, 2001).
³Michael Behe, *Darwin's Black Box* (New York: Free Press, 1996).
⁴Clarification of my use of the word "chance" may be appropriate. In some cases, I use it in the sense of "pure chance" where process is entirely unrestrained. Its use in regard to mutations approaches

this sense, although there are indications that in rare instances there may be "directed mutations." Natural selection, although dependent upon chance events, could be argued as having some direction. The probability calculations for the amino acid sequence of a protein molecule would probably approach "pure chance." For those postulating a designer, applying direction to chance events would become of major importance.

- ⁵E. Melendez-Hevia, T. G. Waddell, and M. Cascante, "The Puzzle of the Citric Acid Cycle," *Journal of Molecular Evolution* 43 (1996): 293-303.
⁶S. M. Messer and S. I. Chan, "Evolution of the Cytochrome *c* Oxidase Proton Pump," *Journal of Molecular Evolution* 46 (1998): 508-20.
⁷*Ibid.*
⁸G. C. Mills, "A Theory of Theistic Evolution as an Alternative to the Naturalistic Theory," *Perspectives on Science and Christian Faith (PSCF)* 47 (1995): 112-22; —, "Theistic Evolution: A Design Theory at the Level of Genetic Information," *Christian Scholar's Review* XXIV (1995): 444-58.
⁹G. C. Mills, "Possible Role of Protein Modules in a Theory of Theistic Evolution," *PSCF* 50 (1998): 136-9; and —, "The Origin of Antibody Diversity," *PSCF* 51 (1999): 254-6.
¹⁰H. P. Yockey, "A Calculation of the Probability of Spontaneous Generation by Information Theory," *Journal of Theoretical Biology* 67 (1977): 377-98.



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