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EVIDENCE FOR DESIGN AT THE FOUNDATION OF LIFE

Urea and Purpose

In the year 1828 the German chemist Friedrich Wöhler heated ammonium cyanate in his laboratory and was amazed to see that urea was produced. Why was he amazed? Because ammonium cyanate is an inorganic chemical—one that does not occur in living organisms. But urea was known to be a biological waste product. Wöhler was the first to demonstrate that a nonliving substance could give rise to a substance produced by living organisms. His experiment shattered the distinction between life and nonlife that was thought to exist up until that time. Moreover, it opened up all of life for scientific study. For if life is made of ordinary matter, the same as rocks and so on, then science can study it. And in the more than 170 years since Wöhler's experiment, science has learned a lot about life. We have discovered the structure of DNA, cracked the genetic code, learned to clone genes, and cells, and even whole organisms.

What has the progress of science told us about the ultimate nature of the universe and life? Well, of course, there are a lot of opinions on the subject, but I think we can break them down into two opposite sides. The first side can perhaps be represented by Richard Dawkins, professor of the public understanding of science at Oxford University. Professor Dawkins has stated that: "The universe we observe has precisely the properties we should expect if there is at bottom no design, no purpose, no evil and no good, nothing but pointless indif-

ference.”¹ Certainly a dreary view, but a seriously proposed one.

The second point of view can be represented by Joseph Cardinal Ratzinger, an advisor to Pope John Paul II. About ten years ago Cardinal Ratzinger wrote a little book entitled *In the Beginning: A Catholic Understanding of the Story of Creation and the Fall*. In the book Cardinal Ratzinger wrote:

Let us go directly to the question of evolution and its mechanisms. Microbiology and biochemistry have brought revolutionary insights here. . . . It is the affair of the natural sciences to explain how the tree of life in particular continues to grow and how new branches shoot out from it. This is not a matter for faith. But we must have the audacity to say that the great projects of the living creation are not the products of chance and error. . . . [They] point to a creating Reason and show us a creating Intelligence, and they do so more luminously and radiantly today than ever before. Thus we can say today with a new certitude and joyousness that the human being is indeed a divine project, which only the creating Intelligence was strong and great and audacious enough to conceive of. Human beings are not a mistake but something willed.²

I would like to make three points about the Cardinal's argument. First, unlike Professor Dawkins, Ratzinger says that nature does appear to exhibit purpose and design. Secondly, to support the argument he points to *physical evidence*—the “great products of the living creation”, which “point to a creating Reason”. Not to philosophical, or theological, or scriptural arguments, but to tangible structures. Thirdly, Ratzinger cites the science of biochemistry—the study of the molecular foundation of life—as having particular relevance to his conclusion. It is my purpose in this essay to show why I think Cardinal Ratzinger has the stronger position, and why Professor Dawkins need not despair.

Explaining the Eye

Of course much of this discussion about the nature of life began in 1859, when Charles Darwin published *The Origin of Species*. In his book Darwin proposed to do what no one had been able to do before him—explain how the great variety and complexity of life might have arisen solely through unguided natural processes. His proposed mechanism was, of course, natural selection acting on random variation. In a nutshell, Darwin recognized that there is variety in all species. Some members of a species are larger than others, some faster, some darker in color. Darwin knew that not all members of a species that are born will survive to reproduce, simply because there is not enough food to sustain them all. And so he reasoned that the ones whose chance variation gave them an edge in the struggle to survive would tend to survive and leave offspring. If the variation could be inherited, then over time the characteristics of the species might change. And over great periods of time, great changes might occur.

Darwin's theory was a very elegant idea. Nonetheless, even in the mid-nineteenth century biologists knew of a number of biological systems that did not appear to be able to be built in the gradual way that Darwin envisioned. One in particular was the eye. Biologists of the time knew that the eye was a very complex structure, containing many components, such as a lens, retina, tear ducts, ocular muscles, and so forth. They knew that if an animal were so unfortunate as to be born without one of the components, the result would be a severe loss of vision or outright blindness. So they doubted that such a system could be put together in the many steps required by natural selection.

Charles Darwin, however, knew about the eye too. And he wrote about it in a section of the *Origin of Species* appropriately entitled “Organs of Extreme Perfection and Complication”, in which he said that he did not really know how the eye might have evolved. Nonetheless, he wrote that if you look at the eyes

of modern organisms, you see considerable variety. In some organisms there really is not an “eye”, but rather just a patch of light-sensitive cells. Now, that arrangement is sufficient for enabling an organism to know if it is in light or darkness, but it does not enable an organism to determine which direction the light is coming from, because light coming from virtually any angle will stimulate the light-sensitive cells. However, Darwin continued, if you take that patch of light-sensitive cells and place it in a small depression, as is seen in some modern animals, light coming from one side will cast a shadow over part of the light-sensitive spot, while the rest is illuminated. In theory such an arrangement could allow the creature to determine which direction the light is coming from. And that would be an improvement. If the cup were deepened, the direction-finding ability would be increased. And if the cup were filled with a gelatinous material, that could be the beginning of a crude lens, a further improvement. Using arguments like these, Darwin was able to convince many of his contemporaries that a gradual evolutionary pathway led from something as simple as a light-sensitive spot to something as complicated as the modern vertebrate eye. And if evolution could explain the eye . . . well, what could it not explain?

But there was a question left unaddressed by Darwin's scheme—where did the light-sensitive spot come from? It seems an odd starting point, since most objects are not light sensitive. Nonetheless, Darwin decided not even to attempt to address the question. He wrote that: “How a nerve comes to be sensitive to light hardly concerns us more than how life itself originated.”³

Well, in the past half-century science has become interested in both those questions: the mechanism of vision and the origin of life. Nonetheless, Darwin was correct, I think, to refuse to address the question, because the science of his day did not have the physical or conceptual tools to begin to investigate it. Just to get a flavor of the science of the mid-nineteenth century, remember that atoms—the basis of all chemistry—were then

considered to be theoretical entities. No one was sure if they really existed. The cell, which we now know to be the basis of life, was thought to be a simple glob of protoplasm, not much more than a microscopic piece of Jell-O. So Darwin refused to address the question and left it as a black box in the hope that future discoveries would vindicate his theory.

“Black box” is a phrase used in science to indicate some machine or system that does something interesting, but no one knows how it works. Its mechanism is unknown because we cannot see inside the box to observe it, or if we can see the workings, they are so complicated that we still do not understand what is going on. For most of us (and certainly for me) a good example of a black box is a computer. I use a computer to process words or play games, but I do not have the foggiest idea how it works. And even if I were to remove the cover and see the inside circuitry, I still could not say how it worked. Well, to scientists of Darwin’s day, the cell was a black box. It did very interesting things, but no one knew how.

When people see a black box in action, they have a psychological tendency to assume that it must be operating by some simple mechanism—the insides of the box must be uncomplicated and working on some easily understood principle. A good example of this tendency was the belief in the spontaneous generation of cellular life from sea mud. In the nineteenth century two prominent scientists and admirers of Darwin—Ernst Haeckel and Thomas Huxley—thought that some mud scraped up by an exploring vessel might be living cells. They could believe this because they thought a cell was, in Haeckel’s words, a “simple little lump of albuminous combination of carbon”.⁴ With the tremendous progress biology has made in this century, of course, we know differently. Now that modern science has opened the black box of the cell, we need to readdress the question that stumped Darwin. What is needed to make a light-sensitive spot? What happens when a photon of light impinges upon a retina?

When a photon first hits the retina, it interacts with a small

organic molecule called 11-cis-retinal.⁵ The shape of retinal is rather bent, but when retinal interacts with the photon, it straightens out, isomerizing into trans-retinal. This is the signal that sets in motion a whole cascade of events resulting in vision. When retinal changes shape, it forces a change in the shape of the protein rhodopsin, which is bound to it. The change in rhodopsin's shape exposes a binding site that allows the protein transducin to stick to it. Now part of the transducin complex dissociates and interacts with a protein called phosphodiesterase. When that happens, the phosphodiesterase acquires the ability chemically to cut a small organic molecule called cyclic-GMP, turning it into 5'-GMP. There is a lot of cyclic-GMP in the cell, and some of it sticks to another protein called an ion channel. Normally the ion channel allows sodium ions into the cell. When the concentration of cyclic-GMP decreases because of the action of the phosphodiesterase, however, the cyclic-GMP bound to the ion channel eventually falls off, causing a change in shape that shuts the channel. As a result, sodium ions can no longer enter the cell, the concentration of sodium in the cell decreases, and the voltage across the cell membrane changes. That in turn causes a wave of electrical polarization to be sent down the optic nerve to the brain. And, when interpreted by the brain, that is vision. So, this is what modern science has discovered about how Darwin's "simple" light-sensitive spot functions.

Darwin's Criterion

Although most people will surely think the above description of the visual cascade is complicated, it is really just a little sketch of the chemistry of vision that ignores a number of things that a functioning visual system actually requires. For instance, I have not discussed the regeneration of the system—how it gets back to the starting point in preparation for the next incoming photon. Nonetheless, I think that the discussion

above is sufficient to show that what Darwin and his contemporaries took as simple starting points have turned out to be enormously complex—much more complex than Darwin ever envisioned.

But how can we tell if the eye and other organisms are too complex to be explained by Darwin's theory? It turns out that Darwin himself gave us a criterion by which to judge his theory. He wrote in the *Origin of Species* that: "If it could be demonstrated that any complex organ existed which could not possibly have been formed by numerous, successive, slight modifications, my theory would absolutely break down."⁶ But what sort of organ or system could not be formed by "numerous, successive, slight modifications"? Well, to begin with, one that is *irreducibly complex*. "Irreducibly complex" is a fancy phrase, but it stands for a very simple concept. As I wrote in *Darwin's Black Box: The Biochemical Challenge to Evolution*, an irreducibly complex system is: "a single system which is composed of several well-matched, interacting parts that contribute to the basic function, and where the removal of any one of the parts causes the system to effectively cease functioning."⁷ Less formally, the phrase "irreducibly complex" just means that a system has a number of components that interact with each other, and if any are taken away the system no longer works. A good illustration of an irreducibly complex system from our everyday world is a simple mechanical mousetrap. The mousetraps that one buys at the hardware store generally have a wooden platform to which all the other parts are attached. It also has a spring with extended ends, one of which presses against the platform, the other against a metal part called the hammer, which actually does the job of squashing the mouse. When one presses the hammer down, it has to be stabilized in that position until the mouse comes along, and that is the job of the holding bar. The end of the holding bar itself has to be stabilized, so it is placed into a metal piece called the catch. All of these pieces are held together by assorted staples.

Now, if the mousetrap is missing the spring, or hammer, or platform, it does not catch mice half as well as it used to, or even a quarter as well. It does not catch mice at all. Therefore it is irreducibly complex. It turns out that irreducibly complex systems are headaches for Darwinian theory, because they are resistant to being produced in the gradual, step-by-step manner that Darwin envisioned. For example, if we wanted to evolve a mousetrap, where would we start? Could we start with just the platform and hope to catch a few mice rather inefficiently? Then add the holding bar, and improve the efficiency a bit? Then add the other pieces one at a time, steadily improving the whole apparatus? No, of course we cannot do that, because the mousetrap does not work at all until it is essentially completely assembled.

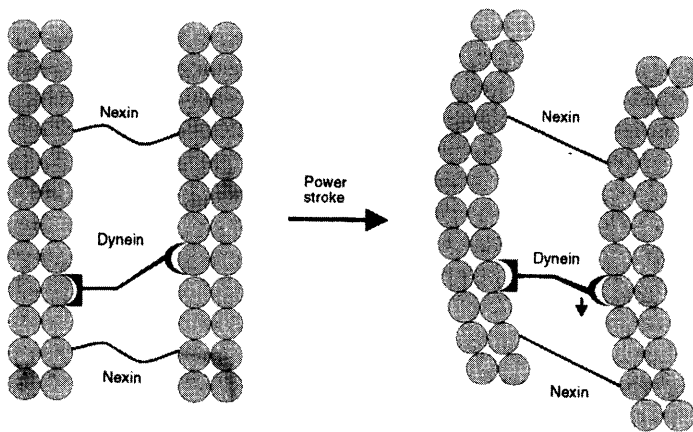
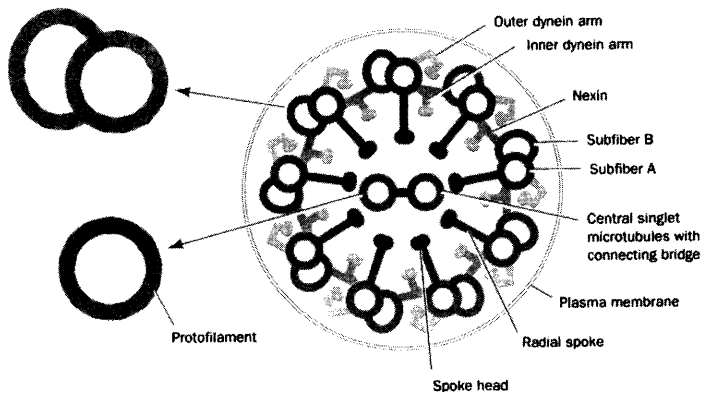
Biochemical Challenges to Darwinism

Mousetraps are one thing, biological systems another. What we really want to know is whether there are any irreducibly complex biological systems, or cellular systems, or biochemical systems. And it turns out that there are many such irreducibly complex systems. Let us consider two examples. The first is called the cilium. A cilium is a little hairlike organelle on the surface of many types of cells. It has the intriguing ability to beat back and forth, moving liquid over the surface of a cell. In some tissue in the lungs, each cell contains hundreds of cilia that beat in synchrony. Interspersed among the ciliated cells are larger ones called goblet cells. The goblet cells secrete mucus into the lining of the lungs, which is swept by the ciliary beating up to the throat where it can be coughed out, along with any dust particles or other foreign objects that might have made their way into the lungs. But what makes a little hairlike organelle beat back and forth? Work in the past several decades has shown that cilia are actually very complicated molecular machines.

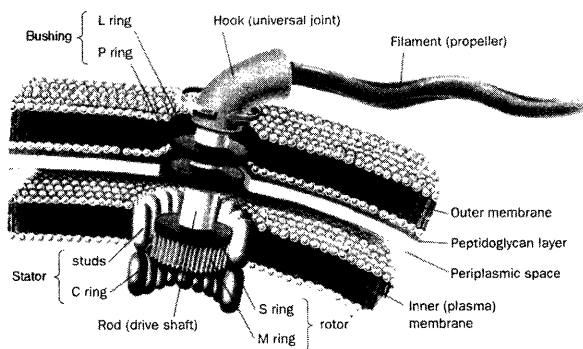
The basic structure of a cilium consists of nine double microtubules.⁸ [See figure on p. 122.] Each of the double microtubules contains two rings made up of ten and thirteen strands respectively of the protein tubulin. In the middle of the cilium are two single microtubules. All of the microtubules are connected to each other by various types of connectors. Neighboring double microtubules are connected by a protein called nexin. The outer double microtubules are connected to the inner single microtubules by radial spokes. And the two inner microtubules are attached by a small connecting bridge. Additionally, on each double microtubule there are two appendages: an outer dynein bridge and an inner dynein bridge. Although this all sounds complicated, such a brief description cannot do justice to the full complexity of the cilium, which, thorough biochemical studies have shown, contains about two hundred different kinds of protein parts.

But how does the cilium work? Studies have shown that it works by a "sliding-fiber mechanism". Neighboring microtubules are the fibers; dynein is a "motor protein". When the cilium is working, the dynein, bound to one strand, reaches over, attaches to a neighboring microtubule, and pushes down. When that happens, the microtubules start to slide with respect to each other. They would continue to slide until they fell apart, except that they are held together by the linker protein nexin. Initially rather loose, as the fibers slide, the nexin becomes more and more taut. As the tension on the nexin and microtubules increases beyond a certain point, the microtubules bend. Thus the sliding motion is converted into a bending motion.

If one thinks about it, it is easy to see that the cilium is irreducibly complex. If it were not for the microtubules, there would be nothing to slide. If the dynein were missing, the whole apparatus would lie stiff and motionless. And if the nexin linkers were missing, the whole apparatus would fall apart when the dynein started to push the microtubules, as it does in experiments when the nexin linkers are removed.



(Top) Cross-section of a cilium showing the fused double-ring structure of the outer microtubules, the single-ring structure of the central microtubules, connecting proteins, and dynein motor. (Bottom) The sliding motion induced by dynein “walking” up a neighboring microtubule is converted to a bending motion by the flexible linker protein nexin. From Voet and Voet, *Biochemistry*, 2d ed. © 1995 John Wiley and Sons, Inc. Reprinted by permission of John Wiley and Sons, Inc.



Drawing of a bacterial flagellum showing the filament, hook, and the motor imbedded in the inner and outer cell membranes and the cell wall. From Voet and Voet, *Biochemistry*, 2d ed. © 1995 John Wiley and Sons, Inc. Reprinted by permission of John Wiley and Sons, Inc.

Much like a mousetrap, a cilium needs a number of parts to function. And, again like a mousetrap, its gradual production in a step-by-step Darwinian fashion is quite difficult to envision.

Another example of an irreducibly complex biochemical system is in some ways like the cilium in that it is an organelle for motion. But in other ways it is completely different. The bacterial flagellum is quite literally an outboard motor that enables some bacteria to swim.⁹ [See figure above.] Like the machines that power our motorboats, the flagellum is a rotary device, in which the rotating surface pushes against the liquid medium, propelling the bacterium along. The part of the flagellum that acts as the propeller is a long whip-like structure made of a protein called flagellin. The propeller is attached to the drive shaft by hook protein, which acts as a universal joint, allowing freedom of rotation for the propeller and drive shaft. The drive shaft is attached to the rotary motor, which uses a flow of acid from outside of the bacterium to the inside in order to power its turning. The drive shaft has to poke through the bacterial

membrane, and several types of proteins act as bushing material to allow that to happen. Although this description makes the flagellum sound complicated, it really does not do justice to its full complexity. Thorough genetic studies have shown that about forty different proteins are required for a functional flagellum, either as parts of the flagellum itself or as parts of the system that builds this machine in the cell. And in the absence of most of those proteins, one does not get a flagellum that spins half as fast as it used to, or a quarter as fast. Either no flagellum gets produced at all, or one that does not work at all. Much like a cilium or mousetrap, the flagellum requires a number of parts to work. Therefore it is irreducibly complex, and its origin presents quite a stumbling block to Darwinian theory.

Darwinian Imagination

I did not discover the cilium or flagellum. It was not I who worked out their mechanisms of action. That work was done by dozens and dozens of laboratories around the world over the course of decades. But if these structures cannot be explained by Darwinian theory, as I contend, then what have other scientists been saying about the origin of molecular machines? One place to look for an answer to that question is in the *Journal of Molecular Evolution*. As its name implies, *JME* was set up specifically to investigate how life might have arisen and then diversified at the molecular level. It is a good journal, which publishes interesting, rigorous material. Of the approximately forty scientists on its editorial board, about fifteen or so are members of the National Academy of Sciences. However, if you pick up a recent copy, you will find that the great majority of papers concern something called "sequence analysis". Briefly, proteins—the components of molecular machines—are made up of "sequences" of amino acids stitched together. Now, if one knows the sequence of amino acids in a protein (or

in its gene) then one can compare the sequence to a similar protein from another species and see where the two sequences are the same, similar, or different. For example, suppose one compared the sequence of the oxygen-carrying protein hemoglobin from a dog to that from a horse. One could then ask, are the amino acid residues in the first position of the two proteins the same or different? How about the second position? the third? the fortieth? And so on. Knowing the answer to that question would be interesting and could indicate how closely related the two species are, and that would be an interesting thing to know.

For our purposes, however, the important point to keep in mind is that comparing sequences does not allow one to conclude how complex molecular machines, such as the cilium or flagellum, could have arisen step by Darwinian step. Perhaps an example would help to show why. Suppose that you compared the bones in the forelimb of a dog to those in the forelimb of a horse. And you observed that there were the same number of bones, and they were arranged in a similar pattern. Knowing that would be interesting, and that might allow you to conclude how closely related the animals are, which again would be an interesting thing to know. However, comparing the bones in the forelimb of a dog to those of a horse will not tell you where bones came from in the first place. In order to do that, you have to build models, do experiments, and so forth. It turns out that virtually none of the papers in the *Journal of Molecular Evolution* over the past decade has done such experimental work or model building.¹⁰ The overwhelming percentage of papers are concerned with sequence analysis. Again, I hasten to say that sequence analysis is interesting and can tell one many things, but sequence analysis alone cannot say how complex molecular machines could have been produced in a Darwinian fashion.

If one looks at other journals, at the *Proceedings of the National Academy of Sciences*, *Cell*, the *Journal of Molecular Biology*, and so on, the story is the same. There are many, many studies

comparing sequences, but very few concerning the Darwinian production of complex molecular machines. The few that do consider the problems of Darwinian evolution are invariably too broad to test rigorously. But if the scientific literature—the journals—do not contain answers to the question of how Darwinian processes could produce such intricate molecular machines, then why do many scientists believe that they can produce them? Well, it is difficult to say in detail, but certainly a part of the answer to that question is that scientists are taught, as part of their scientific training, that Darwinism is true. A good illustration can be found in the excellent textbook *Biochemistry*, by Voet and Voet. In the first chapter, where the textbook is introducing students to the biochemical view of the world, there is a marvelous, full-color drawing depicting the orthodox view of how life arose and diversified. In the top third of the drawing there are illustrated a volcano, lightning flashes, little rays of sunlight, and some gases floating around—and that, students are meant to infer, is how life started. The middle third of the picture shows a stylized drawing of a DNA molecule leading out from the origin-of-life ocean and into a bacterial cell—showing us how life developed. (The bacterium is depicted with a flagellum that, in the far-off view, looks as simple as a hair.) The bottom third of the picture shows the Garden of Eden, with a number of animals produced by evolution milling about. In their midst are a man and woman in the buff (which will no doubt attract student interest). If you look closely you see that the woman is offering the man an apple. And that, students are implicitly led to believe, is how life diversified.

But if you look through the text for serious scientific answers to how any of those processes could have occurred, you will not find them. In the *Origin of Species* at a number of points Darwin appealed to the imagination of his readers. But imagination is a two-edged sword. An imaginative person might see things that other people miss. Or he might see things that are not there. An examination of the science literature seems to

show that Darwinism has become stuck in the world of imagination.

Apprehending Design

My criticisms of Darwinian theory are not really new. A number of other scientists have previously noted that the biochemistry of life is really quite complex and does not seem to fit the gradualistic mechanism that Darwin proposed. Further, it has been pointed out by others that the scientific literature contains few real explanations of the molecular foundations of life. Scientists like Stuart Kauffman of the Santa Fe Institute, James Shapiro of the University of Chicago, and Lynn Margulis of the University of Massachusetts have all stated that natural selection is not a good explanation for some aspects of life.

Where I differ from those other critics is in the alternative I propose. I have written that if you look at molecular machines, such as the cilium, the flagellum, and others, they look like they were designed—purposely designed by an intelligent agent. That proposal has attracted a bit of attention. Some of my critics have pointed out that I am a Roman Catholic and imply therefore that the proposal of intelligent design is a religious idea, not a scientific one. I disagree. I think the conclusion of intelligent design in these cases is completely empirical. That is, it is based entirely on the physical evidence, along with an understanding of how we come to conclude that an object was designed. Every day of our lives we decide, consciously or not, that some things were designed, others not. How do we do that? How do we reach those conclusions?

To begin to see how we conclude that an object or system was designed, imagine that you are walking with a friend in the woods. Suddenly your friend is pulled up by the ankle by a vine and left dangling in the air. After you cut him down, you reconstruct the situation. You see that the vine was tied to a tree limb that was bent down and held by a stake in the ground. The vine was covered by leaves so that you would not

notice it, and so on. From the way the parts were arranged, you would quickly conclude that this was no accident—this was a designed trap. Your conclusion is not based on religious beliefs; it is one based firmly in the physical evidence.

Let us ask a few more questions about the vine-trap. First, who designed it? After reflecting for a minute we see that we do not have enough information to answer that question. Maybe it was an enemy of yours or your friend's; maybe it was a prankster. Without more information we cannot decide who designed the trap. Nonetheless, from the interaction of the parts of the trap, we can conclude that it was indeed designed. A second question is, when was the trap designed? Again, after a minute's thought, we see that we do not yet have enough information to answer the question. Without more data, we cannot decide if the trap was designed an hour ago, a day ago, a week ago, or longer. But again, we apprehend from the interaction of the parts of the trap the fact of design itself. The bottom line is that we need additional information to answer questions such as who, what, where, when, why, and how the trap was designed. But the fact that the trap was designed is apprehended directly from observing the system.

Although we apprehend design easily and intuitively, it can also be treated in an academically rigorous manner. An excellent start has been made in treating the design problem in a philosophically and scientifically rigorous way by the philosopher and mathematician William Dembski in his monograph *The Design Inference: Eliminating Chance through Small Probabilities*.¹¹

In conclusion, I would like to hearken back to the quotations with which I began this essay. In my view there is every reason, based on hard empirical observation, to conclude with Joseph Cardinal Ratzinger that "the great projects of the living creation are not the products of chance and error. . . . [They] point to a creating Reason and show us a creating Intelligence, and they do so more luminously and radiantly today than ever before."

NOTES

¹ G. Easterbrook, "Science and God: A Warming Trend?" *Science* 277 (1997): 890-93.

² J. Ratzinger, *In the Beginning: A Catholic Understanding of the Story of Creation and the Fall* (Grand Rapids, Mich.: Eerdmans, 1986), pp. 54-56.

³ C. Darwin, *On the Origin of Species* (1876; reprint, New York: New York University Press, 1988), p. 151.

⁴ J. Farley, *The Spontaneous Generation Controversy from Descartes to Oparin* (Baltimore: Johns Hopkins University Press, 1977), p. 73.

⁵ T. M. Devlin, *Textbook of Biochemistry* (New York: Wiley-Liss, 1997), chap. 22.3.

⁶ Darwin, *Origin*, p. 154.

⁷ M. J. Behe, *Darwin's Black Box: The Biochemical Challenge to Evolution* (New York: Free Press, 1996), p. 39.

⁸ D. Voet and J. G. Voet, *Biochemistry* (New York: J. Wiley and Sons, 1995), pp. 1252-59.

⁹ *Ibid.*, pp. 1259-60.

¹⁰ Behe, *Darwin's*, chap. 8.

¹¹ W. Dembski, *The Design Inference: Eliminating Chance through Small Probabilities* (Cambridge: Cambridge University Press, 1998).