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Irreducible Complexity

Obstacle to Darwinian Evolution

Michael J. Behe

A SKETCH OF THE INTELLIGENT DESIGN HYPOTHESIS

In his seminal work *On the Origin of Species*, Darwin hoped to explain what no one had been able to explain before – how the variety and complexity of the living world might have been produced by simple natural laws. His idea for doing so was, of course, the theory of evolution by natural selection. In a nutshell, Darwin saw that there was variety in all species. For example, some members of a species are bigger than others, some faster, some brighter in color. He knew that not all organisms that are born will survive to reproduce, simply because there is not enough food to sustain them all. So Darwin reasoned that the ones whose chance variation gives them an edge in the struggle for life would tend to survive and leave offspring. If the variation could be inherited, then over time the characteristics of the species would change, and over great periods of time, perhaps great changes could occur.

It was an elegant idea, and many scientists of the time quickly saw that it could explain many things about biology. However, there remained an important reason for reserving judgment about whether it could actually account for all of biology: the basis of life was as yet unknown. In Darwin's day, atoms and molecules were still theoretical constructs – no one was sure if such things actually existed. Many scientists of Darwin's era took the cell to be a simple glob of protoplasm, something like a microscopic piece of Jell-O. Thus the intricate molecular basis of life was utterly unknown to Darwin and his contemporaries.

In the past hundred years, science has learned much more about the cell and, especially in the past fifty years, much about the molecular basis of life. The discoveries of the double helical structure of DNA, the genetic code, the complicated, irregular structure of proteins, and much else have given us a greater appreciation for the elaborate structures that are necessary to sustain life. Indeed, we have seen that the cell is run by machines – literally,

machines made of molecules. There are molecular machines that enable the cell to move, machines that empower it to transport nutrients, machines that allow it to defend itself.

In light of the enormous progress made by science since Darwin first proposed his theory, it is reasonable to ask if the theory still seems to be a good explanation for life. In *Darwin's Black Box: The Biochemical Challenge to Evolution* (Behe 1996), I argued that it is not. The main difficulty for Darwinian mechanisms is that many systems in the cell are what I termed "irreducibly complex." I defined an irreducibly complex system as: a single system that is necessarily 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 (Behe 2001). As an example from everyday life of an irreducibly complex system, I pointed to a mechanical mousetrap such as one finds in a hardware store. Typically, such traps have a number of parts: a spring, a wooden platform, a hammer, and other pieces. If one removes a piece from the trap, it can't catch mice. Without the spring, or hammer, or any of the other pieces, one doesn't have a trap that works half as well as it used to, or a quarter as well; one has a broken mousetrap, which doesn't work at all.

Irreducibly complex systems seem very difficult to fit into a Darwinian framework, for a reason insisted upon by Darwin himself. In the *Origin*, Darwin wrote that "[i]f 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. But I can find out no such case" (Darwin 1859, 158). Here Darwin was emphasizing that his was a gradual theory. Natural selection had to improve systems by tiny steps, over a long period of time, because if things improved too rapidly, or in large steps, then it would begin to look as if something other than natural selection were driving the process. However, it is hard to see how something like a mousetrap could arise gradually by something akin to a Darwinian process. For example, a spring by itself, or a platform by itself, would not catch mice, and adding a piece to the first nonfunctioning piece wouldn't make a trap either. So it appears that irreducibly complex biological systems would present a considerable obstacle to Darwinian evolution.

The question then becomes, are there any irreducibly complex systems in the cell? Are there any irreducibly complex molecular machines? Yes, there are many. In *Darwin's Black Box*, I discussed several biochemical systems as examples of irreducible complexity: the eukaryotic cilium, the intracellular transport system, and more. Here I will just briefly describe the bacterial flagellum (DeRosier 1998; Shapiro 1995), since its structure makes the difficulty for Darwinian evolution easy to see (Figure 19.1). The flagellum can be thought of as an outboard motor that bacteria use to swim. It was the first truly rotary structure discovered in nature. It consists of a long filamentous

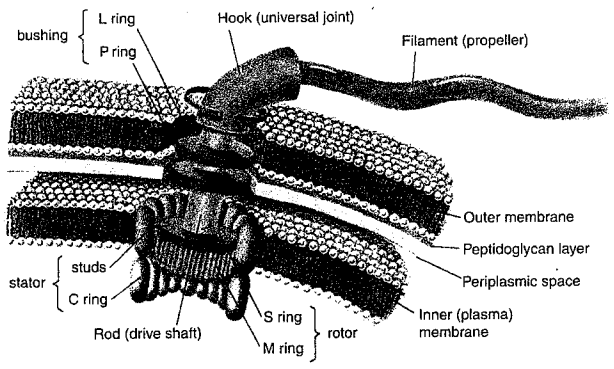

FIGURE 19.1. The bacterial flagellum. Reproduced from D. Voet and J. G. Voet, *Biochemistry*, 2nd ed. (New York: Wiley, 1995), Figure 34–84, with permission of John Wiley Publishers and Donald Voet, who wished to emphasize that "this is an artist-drawn representation of the flagellum rather than a photo or drawing of an actual flagellum."

tail that acts as a propeller; when it is spun, it pushes against the liquid medium and can propel the bacterium forward. The propeller is attached to the drive shaft indirectly through something called the hook region, which acts as a universal joint. The drive shaft is attached to the motor, which uses a flow of acid or sodium ions from the outside to the inside of the cell to power rotation. Just as an outboard motor has to be kept stationary on a motorboat while the propeller turns, there are proteins that act as a stator structure to keep the flagellum in place. Other proteins act as bushings to permit the drive shaft to pass through the bacterial membrane. Studies have shown that thirty to forty proteins are required to produce a functioning flagellum in the cell. About half of the proteins are components of the finished structure, while the others are necessary for the construction of the flagellum. In the absence of almost any of the proteins – in the absence of the parts that act as the propeller, drive shaft, hook, and so forth – no functioning flagellum is built.

As with the mousetrap, it is quite difficult to see how Darwin's gradualistic process of natural selection sifting random mutations could produce the bacterial flagellum, since many pieces are required before its function appears. A hook by itself, or a driveshaft by itself, will not act as a propulsive device. But the situation is actually much worse than it appears from this cursory description, for several reasons. First, there is associated with the functioning of the flagellum an intricate control system, which tells the flagellum when to rotate, when to stop, and sometimes when to reverse itself

and rotate in the opposite direction. This allows the bacterium to swim toward or away from an appropriate signal, rather than in a random direction that could much more easily take it the wrong way. Thus the problem of accounting for the origin of the flagellum is not limited to the flagellum itself but extends to associated control systems as well.

Second, a more subtle problem is how the parts assemble themselves into a whole. The analogy to an outboard motor fails in one respect: an outboard motor is generally assembled under the direction of a human – an intelligent agent who can specify which parts are attached to which other parts. The information for assembling a bacterial flagellum, however (or, indeed, for assembling any biomolecular machine), resides in the component proteins of the structure itself. Recent work shows that the assembly process for a flagellum is exceedingly elegant and intricate (Yonekura et al. 2000). If that assembly information is absent from the proteins, then no flagellum is produced. Thus, even if we had a hypothetical cell in which proteins homologous to all of the parts of the flagellum were present (perhaps performing jobs other than propulsion) but were missing the information on how to assemble themselves into a flagellum, we would still not get the structure. The problem of irreducibility would remain.

Because of such considerations, I have concluded that Darwinian processes are not promising explanations for many biochemical systems in the cell. Instead, I have noted that, if one looks at the interactions of the components of the flagellum, or cilium, or other irreducibly complex cellular system, they look like they were designed – purposely designed by an intelligent agent. The features of the systems that indicate design are the same ones that stymie Darwinian explanations: the specific interaction of multiple components to accomplish a function that is beyond the individual components. The logical structure of the argument to design is a simple inductive one: whenever we see such highly specific interactions in our everyday world, whether in a mousetrap or elsewhere, we unfailingly find that the systems were intentionally arranged – that they were designed. Now we find systems of similar complexity in the cell. Since no other explanation has successfully addressed them, I argue that we should extend the induction to subsume molecular machines, and hypothesize that they were purposely designed.

MISCONCEPTIONS ABOUT WHAT A HYPOTHESIS OF DESIGN ENTAILS

The hypothesis of Intelligent Design (ID) is quite controversial, mostly because of its philosophical and theological overtones, and in the years since *Darwin's Black Box* was published a number of scientists and philosophers have tried to refute its main argument. I have found these rebuttals to be unpersuasive, at best. Quite the opposite, I think that some putative

counterexamples to design are unintentionally instructive. Not only do they fail to make their case for the sufficiency of natural selection, they show clearly the obstacle that irreducible complexity poses to Darwinism. They also show that Darwinists have great trouble recognizing problems with their own theory. I will examine two of those counterexamples in detail a little later in this chapter. Before I do, however, I will first address a few common misconceptions that surround the biochemical design argument.

First of all, it is important to understand that a hypothesis of Intelligent Design has no quarrel with evolution per se – that is, evolution understood simply as descent with modification, but leaving the mechanism open. After all, a designer may have chosen to work that way. Rather than common descent, the focus of ID is on the *mechanism* of evolution – how did all this happen, by natural selection or by purposeful Intelligent Design?

A second point that is often overlooked but should be emphasized is that Intelligent Design can happily coexist with even a large degree of natural selection. Antibiotic and pesticide resistance, antifreeze proteins in fish and plants, and more may indeed be explained by a Darwinian mechanism. The critical claim of ID is not that natural selection doesn't explain *anything*, but that it doesn't explain *everything*.

My book, *Darwin's Black Box*, in which I flesh out the design argument, has been widely discussed in many publications. Although many issues have been raised, I think the general reaction of scientists to the design argument is well and succinctly summarized in the recent book *The Way of the Cell*, published by Oxford University Press and authored by the Colorado State University biochemist Franklin Harold. Citing my book, Harold writes, "We should reject, as a matter of principle, the substitution of intelligent design for the dialogue of chance and necessity (Behe 1996); but we must concede that there are presently no detailed Darwinian accounts of the evolution of any biochemical system, only a variety of wishful speculations" (Harold 2001, 205).

Let me emphasize, in reverse order, Harold's two points. First, as other reviewers of my book have done,[1] Harold acknowledges that Darwinists have no real explanation for the enormous complexity of the cell, only hand-waving speculations, more colloquially known as "just-so stories." I had claimed essentially the same thing six years earlier in *Darwin's Black Box* and encountered fierce resistance – mostly from internet fans of Darwinism who claimed that, why, there were hundreds or thousands of research papers describing the Darwinian evolution of irreducibly complex biochemical systems, and who set up web sites to document them.[2]

As a sufficient response to such claims, I will simply rely on Harold's statement quoted here, as well as the other reviewers who agree that there is a dearth of Darwinian explanations. After all, if prominent scientists who are no fans of Intelligent Design agree that the systems remain unexplained, then that should settle the matter. Let me pause, however, to note that I find

this an astonishing admission for a theory that has dominated biology for so long. That Darwinian theory has borne so little fruit in explaining the molecular basis of life – despite its long reign as the fundamental theory of biology – strongly suggests that it is not the right framework for understanding the origin of the complexity of life.

Harold's second point is that there is some principle that forbids us from investigating Intelligent Design, even though design is an obvious idea that quickly pops into your mind when you see a drawing of the flagellum (Figure 19.1) or other complex biochemical system. What principle is that? He never spells it out, but I think the principle probably boils down to this: design appears to point strongly beyond nature. It has philosophical and theological implications, and that makes many people uncomfortable. Because they think that science should avoid a theory that points so strongly beyond nature, they want to rule out intelligent design from the start.

I completely disagree with that view and find it fainthearted. I think science should follow the evidence wherever it seems to lead. That is the only way to make progress. Furthermore, not only Intelligent Design, but *any* theory that purports to explain how life occurred will have philosophical and theological implications. For example, the Oxford biologist Richard Dawkins has famously said that "Darwin made it possible to be an intellectually-fulfilled atheist" (Dawkins 1986, 6). A little less famously, Kenneth Miller has written that "[God] used evolution as the tool to set us free" (Miller 1999, 253). Stuart Kauffman, a leading complexity theorist, thinks Darwinism cannot explain all of biology: "Darwinism is not enough. . . . [N]atural selection cannot be the sole source of order we see in the world" (Kauffman 1995, viii). But Kauffman thinks that his theory will somehow show that we are "at home in the universe." The point, then, is that all theories of origins carry philosophical and theological implications. There is no way to avoid them in an explanation of life.

Another source of difficulty for some people concerns the question, how could biochemical systems have been designed? A common misconception is that designed systems would have to be created from scratch in a puff of smoke. But that isn't necessarily so. The design process may have been much more subtle. In fact, it may have contravened no natural laws at all. Let's consider just one possibility. Suppose the designer is indeed God, as most people would suspect. Well, then, as Kenneth Miller points out in his book, *Finding Darwin's God*:

> The indeterminate nature of quantum events would allow a clever and subtle God to influence events in ways that are profound, but scientifically undetectable to us. Those events could include the appearance of mutations . . . and even the survival of individual cells and organisms affected by the chance processes of radioactive decay. (Miller 1999, 241)

Miller doesn't think that guidance is necessary in evolution, but if it were (as I believe), then a route would be open for a subtle God to design life without overriding natural law. If quantum events such as radioactive decay are not governed by causal laws, then it breaks no law of nature to influence such events. As a theist like Miller, that seems perfectly possible to me. I would add, however, that such a process would amount to Intelligent Design, not Darwinian evolution. Further, while we might not be able to detect quantum manipulations, we may nevertheless be able to conclude confidently that the final structure was designed.

MISCONCEPTIONS CONCERNING SUPPOSED WAYS AROUND THE IRREDUCIBILITY OF BIOCHEMICAL SYSTEMS

Consider a hypothetical example where proteins homologous to all of the parts of an irreducibly complex molecular machine first had other individual functions in the cell. Might the irreducible system then have been put together from individual components that originally worked on their own, as some Darwinists have proposed? Unfortunately, this picture greatly oversimplifies the difficulty, as I discussed in *Darwin's Black Box* (Behe 1996, 53). Here analogies to mousetraps break down somewhat, because the parts of a molecular system have to find each other automatically in the cell. They can't be arranged by an intelligent agent, as a mousetrap is. In order to find each other in the cell, interacting parts have to have their surfaces shaped so that they are very closely matched to each other, as pictured in Figure 19.2. Originally, however, the individually acting components would not have had complementary surfaces. So all of the interacting surfaces of

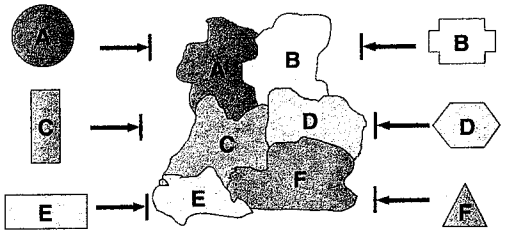

FIGURE 19.2. The parts of an irreducibly complex molecular machine must have surfaces that are closely matched to each other to allow specific binding. This drawing emphasizes that even if individually acting proteins homologous to parts of a complex originally had separate functions, their surfaces would not be complementary to each other. Thus the problem of irreducibility remains even if the separate parts originally had individual functions. (The blocked arrows indicate that the original protein shapes are not suitable to bind other proteins in the molecular machine.)

all of the components would first have to be adjusted before they could function together. And only then would the new function of the composite system appear. Thus, I emphasize strongly, *the problem of irreducibility remains, even if individual proteins homologous to system components separately and originally had their own functions.*

Another area where one has to be careful is in noticing that some systems that have extra or redundant components may have an irreducibly complex *core.* For example, a car with four spark plugs might get by with three or two, but it certainly can't get by with none. Rat traps often have two springs, to give them extra strength. The trap can still work if one spring is removed, but it can't work if both springs are removed. Thus in trying to imagine the origin of a rat trap by Darwinian means, we still have all the problems we had with a mousetrap. A cellular example of redundancy is the hugely complex eukaryotic cilium, which contains about 250 distinct protein parts (Dutcher 1995). The cilium has multiple copies of a number of components, including multiple microtubules and dynein arms. Yet a working cilium needs at least one copy of each in order to work, as I pictured in my book (Behe 1996, 60). Thus, like the rat trap's, its gradual Darwinian production remains quite difficult to envision. Kenneth Miller has pointed to the redundancy of the cilium as a counterexample to my claim of its irreducibility (Miller 1999, 140–3). But redundancy only delays irreducibility; it does not eliminate it.

Finally, rather than showing how their theory could handle the obstacle, some Darwinists are hoping to get around irreducible complexity by verbal tap dancing. At a debate between proponents and opponents of Intelligent Design sponsored by the American Museum of Natural History in April 2002, Kenneth Miller actually claimed (the transcript is available at the web site of the National Center for Science Education) that a mousetrap isn't irreducibly complex because subsets of a mousetrap, and even each individual part, could still "function" on their own. The holding bar of a mousetrap, Miller observed, could be used as *a toothpick,* so it still has a "function" outside the mousetrap. Any of the parts of the trap could be used as a paperweight, he continued, so they all have "functions." And since any object that has mass can be a paperweight, then any part of anything has a function of its own. *Presto,* there is no such thing as irreducible complexity! Thus the acute problem for gradualism that any child can see in systems like the mousetrap is smoothly explained away.

Of course, the facile explanation rests on a transparent fallacy, a brazen equivocation. Miller uses the word "function" in two different senses. Recall that the definition of irreducible complexity notes that removal of a part "causes the *system* to effectively cease functioning." Without saying so, in his exposition Miller shifts the focus from the separate function of the intact *system* itself to the question of whether we can find a different use (or "function") for some of the *parts.* However, if one removes a part from the mousetrap that I have pictured, it can no longer catch mice. The *system* has

indeed effectively ceased functioning, so the *system* is irreducibly complex, just as I have written. What's more, the functions that Miller glibly assigns to the parts – paperweight, toothpick, key chain, and so forth – have little or nothing to do with the function of the system – catching mice (unlike the mousetrap series proposed by John McDonald, to be discussed later) – so they give us no clue as to how the system's function could arise gradually. Miller has explained precisely nothing.

With the problem of the mousetrap behind him, Miller then moved on to the bacterial flagellum – and again resorted to the same fallacy. If nothing else, one has to admire the breathtaking audacity of verbally trying to turn another severe problem for Darwinism into an advantage. In recent years, it has been shown that the bacterial flagellum is an even more sophisticated system than had been thought. Not only does it act as a rotary propulsion device, it also contains within itself an elegant mechanism used to transport the proteins that make up the outer portion of the machine from the inside of the cell to the outside (Aizawa 1996). Without blinking, Miller asserted that the flagellum is not irreducibly complex because some proteins of the flagellum could be missing and the remainder could still transport proteins, perhaps independently. (Proteins similar – but not identical – to some found in the flagellum occur in the type III secretory system of some bacteria. See Hueck 1998). Again, he was equivocating, switching the focus from the function of the system, acting as a rotary propulsion machine, to the ability of a subset of the system to transport proteins across a membrane. However, taking away the parts of the flagellum certainly destroys the ability of the system to act as a rotary propulsion machine, as I have argued. Thus, contra Miller, the flagellum is indeed irreducibly complex. What's more, the function of transporting proteins has as little directly to do with the function of rotary propulsion as a toothpick has to do with a mousetrap. So discovering the supportive function of transporting proteins tells us precisely nothing about how Darwinian processes might have put together a rotary propulsion machine.

THE BLOOD CLOTTING CASCADE

Having dealt with some common misconceptions about intelligent design, in the next two sections I will examine two systems that were proposed as serious counterexamples to my claim of irreducible complexity. I will show not only that they fail, but also how they highlight the seriousness of the obstacle of irreducible complexity.

In *Darwin's Black Box*, I argued that the blood clotting cascade is an example of an irreducibly complex system (Behe 1996, 74–97). At first glance, clotting seems to be a simple process. A small cut or scrape will bleed for a while and then slow down and stop as the visible blood congeals. However, studies over the past fifty years have shown that the visible simplicity is

undergirded by a system of remarkable complexity (Halkier 1992). In all, there are over a score of separate protein parts involved in the vertebrate clotting system. The concerted action of the components results in the formation of a weblike structure at the site of the cut, which traps red blood cells and stops the bleeding. Most of the components of the clotting cascade are involved not in the structure of the clot itself, but in the control of the timing and placement of the clot. After all, it would not do to have clots forming at inappropriate times and places. A clot that formed in the wrong place, such as in the heart or brain, could lead to a heart attack or stroke. Yet a clot that formed even in the right place, but too slowly, would do little good.

The insoluble weblike fibers of the clot material itself are formed of a protein called fibrin. However, an insoluble web would gum up blood flow before a cut or scrape happened, so fibrin exists in the bloodstream initially in a soluble, inactive form called fibrinogen. When the closed circulatory system is breached, fibrinogen is activated by having a piece cut off from one end of two of the three proteins that comprise it. This exposes sticky sites on the protein, which allows them to aggregate. Because of the shape of the fibrin, the molecules aggregate into long fibers that form the meshwork of the clot. Eventually, when healing is completed, the clot is removed by an enzyme called plasmin.

The enzyme that converts fibrinogen to fibrin is called thrombin. Yet the action of thrombin itself has to be carefully regulated. If it were not, then thrombin would quickly convert fibrinogen to fribrin, causing massive blood clots and rapid death. It turns out that thrombin exists in an inactive form called prothrombin, which has to be activated by another component called Stuart factor. But by the same reasoning, the activity of Stuart factor has to be controlled, too, and it is activated by yet another component. Ultimately, the component that usually begins the cascade is tissue factor, which occurs on cells that normally do not come in contact with the circulatory system. However, when a cut occurs, blood is exposed to tissue factor, which initiates the clotting cascade.

Thus in the clotting cascade, one component acts on another, which acts on the next, and so forth. I argued that the cascade is irreducibly complex because, if a component is removed, the pathway is either immediately turned on or permanently turned off. It would not do, I wrote, to postulate that the pathway started from one end, fibrinogen, and then added components, since fibrinogen itself does no good. Nor is it plausible even to start with something like fibrinogen and a nonspecific enzyme that might cleave it, since the clotting would not be regulated and would be much more likely to do harm than good.

So said I. But Russell Doolittle – an eminent protein biochemist, a professor of biochemistry at the University of California–San Diego, a member of the National Academy of Sciences, and a lifelong student of the blood

clotting system – disagreed. As part of a symposium discussing my book and Richard Dawkins' *Climbing Mount Improbable* in the *Boston Review*, which is published by the Massachusetts Institute of Technology, Doolittle wrote an essay discussing the phenomenon of gene duplication – the process by which a cell may be provided with an extra copy of a functioning gene. He then conjectured that the components of the blood clotting pathway, many of which have structures that are similar to each other, arose by gene duplication and gradual divergence. This is the common view among Darwinists. Professor Doolittle went on to describe a then-recent experiment that, he thought, showed that the cascade is not irreducible after all. Professor Doolittle cited a paper by Bugge and colleagues (1996a) entitled "Loss of Fibrinogen Rescues Mice from the Pleiotropic Effects of Plasminogen Deficiency." Of that paper, he wrote:

> Recently the gene for plaminogen [sic] was knocked out of mice, and, predictably, those mice had thrombotic complications because fibrin clots could not be cleared away. Not long after that, the same workers knocked out the gene for fibrinogen in another line of mice. Again, predictably, these mice were ailing, although in this case hemorrhage was the problem. And what do you think happened when these two lines of mice were crossed? For all practical purposes, the mice lacking both genes were normal! Contrary to claims about irreducible complexity, the entire ensemble of proteins is not needed. Music and harmony can arise from a smaller orchestra. (Doolittle 1997)

(Again, fibrinogen is the precursor of the clot material itself. Plasminogen is the precursor of plasmin, which removes clots once their purpose is accomplished.) So if one knocks out either one of those genes of the clotting pathway, trouble results; but, Doolittle asserted, if one knocks out both, then the system is apparently functional again. That would be a very interesting result, but it turns out to be incorrect. Doolittle misread the paper.

The abstract of the paper states that "[m]ice deficient in plasminogen and fibrinogen are phenotypically indistinguishable from fibrinogen-deficient mice." In other words, the double mutants have all the problems that the mice lacking just fibrinogen have. Those problems include inability to clot, hemorrhaging, and death of females during pregnancy. Plasminogen deficiency leads to a different suite of symptoms – thrombosis, ulcers, and high mortality. Mice missing both genes were "rescued" from the ill effects of plasminogen deficiency only to suffer the problems associated with fibrinogen deficiency.[3] The reason for this is easy to see. Plasminogen is needed to remove clots that, left in place, interfere with normal functions. However, if the gene for fibrinogen is also knocked out, then clots can't form in the first place, and their removal is not an issue. Yet if clots can't form, then there is no functioning clotting system, and the mice suffer the predictable consequences.

TABLE 19.1. *Effects of knocking out genes for blood clotting components*

Missing Protein	Symptoms	Reference
Plasminogen	Thrombosis, high mortality	Bugge et al. 1995
Fibrinogen	Hemorrhage, death in pregnancy	Suh et al. 1995
Plasminogen/fibrinogen	Hemorrhage, death in pregnancy	Bugge et al. 1996a
Prothrombin	Hemorrhage, death in pregnancy	Sun et al. 1998
Tissue factor	Hemorrhage, death in pregnancy	Bugge et al. 1996b

Clearly, the double-knockout mice are not "normal." They are not promising evolutionary intermediates.

The same group that produced the mice missing plasminogen and fibrinogen has also produced mice individually missing other components of the clotting cascade – prothrombin and tissue factor. In each case, the mice are severely compromised, which is *exactly* what one would expect if the cascade is irreducibly complex (Table 19.1).

What lessons can we draw from this incident? The point is certainly not that Russell Doolittle misread a paper, which anyone might do. (Scientists, as a rule, are not known for their ability to write clearly, and Bugge and colleagues were no exception.) Rather, the main lesson is that irreducible complexity seems to be a much more severe problem than Darwinists recognize, since the experiment Doolittle himself chose to demonstrate that "music and harmony can arise from a smaller orchestra" showed exactly the opposite. A second lesson is that gene duplication is not the panacea that it is often made out to be. Professor Doolittle knows as much about the structures of the clotting proteins and their genes as anyone on Earth, and he is convinced that many of them arose by gene duplication and exon shuffling. Yet that knowledge did not prevent him from proposing utterly nonviable mutants as possible examples of evolutionary intermediates. A third lesson is that, as I had claimed in *Darwin's Black Box*, there are no papers in the scientific literature detailing how the clotting pathway could have arisen by Darwinian means. If there were, Doolittle would simply have cited them.

Another significant lesson that we can draw is that, while the majority of academic biologists and philosophers place their confidence in Darwinism, that confidence rests on no firmer grounds than Professor Doolittle's. As an illustration, consider the words of the philosopher Michael Ruse:

> For example, Behe is a real scientist, but this case for the impossibility of a small-step natural origin of biological complexity has been trampled upon contemptuously by the scientists working in the field. They think his grasp of the pertinent science is weak and his knowledge of the literature curiously (although conveniently) outdated.

For example, far from the evolution of clotting being a mystery, the past three decades of work by Russell Doolittle and others has thrown significant light on the ways in which clotting came into being. More than this, it can be shown that the clotting mechanism does not have to be a one-step phenomenon with everything already in place and functioning. One step in the cascade involves fibrinogen, required for clotting, and another, plaminogen [*sic*], required for clearing clots away. (Ruse 1998)

And Ruse goes on to quote Doolittle's passage from the *Boston Review* that I quoted earlier. Now, Ruse is a prominent Darwinist and has written many books on various aspects of Darwiniana. Yet, as his approving quotation of Doolittle's mistaken reasoning shows (complete with his copying of Doolittle's typo-misspelling of "plaminogen"), Ruse has no independent knowledge of how natural selection could have put together complex biochemical systems. As far as the scientific dispute is concerned, Ruse has nothing to add.

Another such example is seen in a recent essay in *The Scientist*, "Not-So-Intelligent Design," by Neil S. Greenspan, a professor of pathology at Case Western Reserve University, who writes (Greenspan 2002), "The Design advocates also ignore the accumulating examples of the reducibility of biological systems. As Russell Doolittle has noted in commenting on the writings of one ID advocate . . ." Greenspan goes on to cite approvingly Doolittle's argument in the *Boston Review*. He concludes, with unwitting irony, that "[t]hese results cast doubt on the claim by proponents of ID that they know which systems exhibit irreducible complexity and which do not." But since the results are precisely the opposite of what Greenspan supposed, the shoe is now on the other foot. This incident casts grave doubt on the claim by Darwinists – both biologists and philosophers – that they know that complex cellular systems are explainable in Darwinian terms. It demonstrates that Darwinists either cannot or will not recognize difficulties for their theory.

THE MOUSETRAP

The second counterargument to irreducibility I will discuss here concerns not a biological example but a conceptual one. In *Darwin's Black Box*, I pointed to a common mechanical mousetrap as an example of irreducible complexity. Almost immediately after the book's publication, some Darwinists began proposing ways in which the mousetrap could be built step by step. One proposal that has gotten wide attention, and that has been endorsed by some prominent scientists, was put forward by John McDonald, a professor of biology at the University of Delaware, and can be seen on his web site.[4] His series of traps is shown in Figure 19.3. McDonald's main point was that the trap that I pictured in my book consisted of five parts, yet he could build a trap with fewer parts.

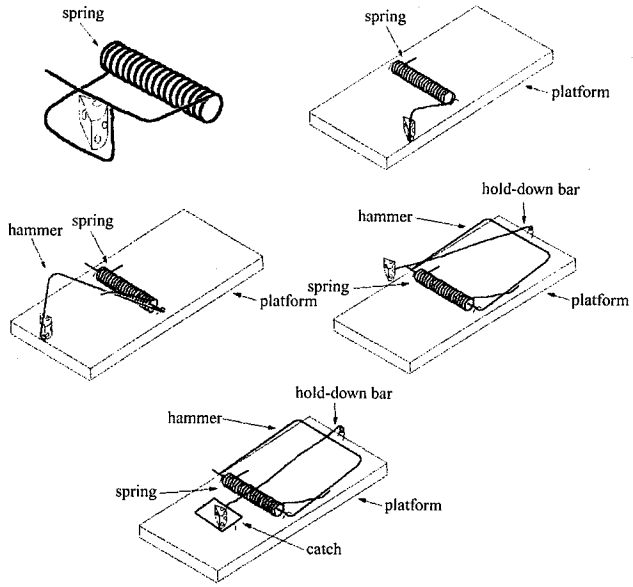

FIGURE 19.3. A series of mousetraps with an increasing number of parts, as proposed by John McDonald <http://udel.edu/~mcdonald/oldmousetrap.html> and reproduced here with his permission. Yet intelligence is still required to construct one trap from another, as described in the text.

I agree. In fact, I said exactly the same thing in my book. I wrote:

We need to distinguish between a *physical* precursor and a *conceptual* precursor. The trap described above is not the only system that can immobilize a mouse. On other occasions my family has used a glue trap. In theory at least, one can use a box propped open with a stick that could be tripped. Or one can simply shoot the mouse with a BB gun. However, these are not physical precursors to the standard mousetrap since they cannot be transformed, step-by-Darwinian-step, into a trap with a base, hammer, spring, catch, and holding bar. (Behe 1996, 43)

Thus the point is not that mousetraps can be built in different ways, with different numbers of pieces. (My children have a game at home called "Mousetrap," which has many, many pieces and looks altogether different from the common mechanical one.) Of course they can. The only question is whether a particular trap can be built by "numerous, successive, slight modifications"

to a simple starting point – without the intervention of intelligence – as Darwin insisted that his theory required.

The McDonald traps cannot. Shown at the top of Figure 19.3 are his one-piece trap and his two-piece trap. The structure of the second trap, however, is not a single, small, random step away from the first. First notice that the one-piece trap is not a simple spring – it is shaped in a very special way. In fact, the shape was deliberately chosen by an intelligent agent, John McDonald, to act as a trap. Well, one has to start somewhere. But if the mousetrap series is to have any relevance at all to Darwinian evolution, then intelligence can't be involved at any further point.

Yet intelligence saturates the whole series. Consider what would be necessary to convert the one-piece trap to the "two-piece" trap. One can't just place the first trap on a simple piece of wood and have it work as the second trap does. Rather, as shown in Figure 19.3, the two protruding ends of the spring first have to be reoriented. What's more, two staples (barely visible in Figure 19.3) are added to hold the spring onto the platform so that it can be under tension in the two-piece trap. So we have gone not from a one-piece to a two-piece trap, but from a one-piece to a four-piece trap. Notice also that the placement of the staples in relation to the edge of the platform is critical. If the staples were moved a quarter-inch from where they are, the trap wouldn't work. Finally, consider that, in order to have a serious analogy to the robotic processes of the cell, we can't have an intelligent human setting the mousetrap – the first trap would have to be set by some unconscious charging mechanism. So, when the pieces are rearranged, the charging mechanism too would have to change for the second trap.

It's easy for us intelligent agents to overlook our role in directing the construction of a system, but nature cannot overlook any step at all, so the McDonald mousetrap series completely fails as an analogy to Darwinian evolution. In fact, the second trap is best viewed not as some Darwinian descendant of the first but as a completely different trap, designed by an intelligent agent, perhaps using a refashioned part or two from the first trap.

Each of the subsequent steps in the series suffers from analogous problems, which I have discussed elsewhere.[5]

In his endorsement of the McDonald mousetrap series, Kenneth Miller wrote: "If simpler versions of this mechanical device [the mousetrap] can be shown to work, then simpler versions of biochemical machines could work as well . . . and this means that complex biochemical machines could indeed have had functional precursors."[6] But that is exactly what it doesn't show – if by "precursor" Miller means "Darwinian precursor." On the contrary, McDonald's mousetrap series shows that even if one does find a simpler system to perform some function, that gives one no reason to think that a more complex system performing the same function could be produced by a Darwinian process starting with the simpler system. Rather, the difficulty in doing so for a simple mousetrap gives us compelling reason to think it cannot be done for complex molecular machines.

FUTURE PROSPECTS OF THE INTELLIGENT DESIGN HYPOTHESIS

The misconceived arguments by Darwinists that I have recounted here offer strong encouragement to me that the hypothesis of Intelligent Design is on the right track. After all, if well-informed opponents of an idea attack it by citing data that, when considered objectively, actually demonstrate its force, then one is entitled to be confident that the idea is worth investigating.

Yet it is not primarily the inadequacy of Darwinist responses that bodes well for the design hypothesis. Rather, the strength of design derives mainly from the work-a-day progress of science. In order to appreciate this fact, it is important to realize that the idea of Intelligent Design arose not from the work of any individual but from the collective work of biology, particularly in the last fifty years. Fifty years ago, the cell seemed much simpler, and in our innocence it was easier then to think that Darwinian processes might have accounted for it. But as biology progressed and the imagined simplicity vanished, the idea of design became more and more compelling. That trend is continuing inexorably. The cell is not getting any simpler; it is getting much more complex. I will conclude this chapter by citing just one example, from the relatively new area of proteomics.

With the successful sequencing of the entire genomes of dozens of microorganisms and one vertebrate (us), the impetus has turned toward analyzing the cellular interactions of the proteins that the genomes code for, taken as a whole. Remarkable progress has already been made. Early in 2002, an exhaustive study of the proteins comprising the yeast proteome was reported. Among other questions, the investigators asked what proportion of yeast proteins work as groups. They discovered that nearly fifty percent of proteins work as complexes of a half-dozen or more, and many as complexes of ten or more (Gavin et al. 2002).

This is not at all what Darwinists had expected. As Bruce Alberts wrote earlier in the article "The Cell as a Collection of Protein Machines":

> We have always underestimated cells. Undoubtedly we still do today. But at least we are no longer as naive as we were when I was a graduate student in the 1960s. Then most of us viewed cells as containing a giant set of second-order reactions. . . .
>
> But, as it turns out, we can walk and we can talk because the chemistry that makes life possible is much more elaborate and sophisticated than anything we students had ever considered. Proteins make up most of the dry mass of a cell. But instead of a cell dominated by randomly colliding individual protein molecules, we now know that nearly every major process in a cell is carried out by assemblies of 10 or more protein molecules. And, as it carries out its biological functions, each of these protein assemblies interacts with several other large complexes of proteins.

Indeed, the entire cell can be viewed as a factory that contains an elaborate network of interlocking assembly lines, each of which is composed of a set of large protein machines. (Alberts 1998)

The important point here for a theory of Intelligent Design is that molecular machines are not confined to the few examples that I discussed in *Darwin's Black Box.* Rather, most proteins are found as components of complicated molecular machines. Thus design might extend to a large fraction of the features of the cell, and perhaps beyond that into higher levels of biology.

Progress in twentieth-century science has led us to the design hypothesis. I expect progress in the twenty-first century to confirm and extend it.

Notes

1. For example, the microbiologist James Shapiro of the University of Chicago declared in *National Review* that "[t]here are no detailed Darwinian accounts for the evolution of any fundamental biochemical or cellular system, only a variety of wishful speculations" (Shapiro 1996, 65). In *Nature,* the University of Chicago evolutionary biologist Jerry Coyne stated, "There is no doubt that the pathways described by Behe are dauntingly complex, and their evolution will be hard to unravel.... [W]e may forever be unable to envisage the first proto-pathways" (Coyne 1996, 227). In a particularly scathing review in *Trends in Ecology and Evolution,* Tom Cavalier-Smith, an evolutionary biologist at the University of British Columbia, nonetheless wrote, "For none of the cases mentioned by Behe is there yet a comprehensive and detailed explanation of the probable steps in the evolution of the observed complexity. The problems have indeed been sorely neglected – though Behe repeatedly exaggerates this neglect with such hyperboles as 'an eerie and complete silence' " (Cavalier-Smith 1997, 162). The Evolutionary biologist Andrew Pomiankowski, writing in *New Scientist,* agreed: "Pick up any biochemistry textbook, and you will find perhaps two or three references to evolution. Turn to one of these and you will be lucky to find anything better than 'evolution selects the fittest molecules for their biological function' " (Pomiankowski 1996, 44). In *American Scientist,* the Yale molecular biologist Robert Dorit averred, "In a narrow sense, Behe is correct when he argues that we do not yet fully understand the evolution of the flagellar motor or the blood clotting cascade" (Dorit 1997, 474).
2. A good example is found on the "World of Richard Dawkins" web site, maintained by a Dawkins fan named John Catalano at <www.world-of-dawkins.com/Catalano/box/published.htm>. It is to this site that the Oxford University physical chemist Peter Atkins was referring when he wrote in a review of *Darwin's Black Box* for the "Infidels" web site: "Dr. Behe claims that science is largely silent on the details of molecular evolution, the emergence of complex biochemical pathways and processes that underlie the more traditional manifestations of evolution at the level of organisms. Tosh! There are hundreds, possibly thousands, of scientific papers that deal with this very subject. For an entry into this important and flourishing field, and an idea of the intense scientific effort that it represents (see the first link above) [*sic*]" (Atkins 1998).

3. Bugge and colleagues (1996a) were interested in the question of whether plasminogen had any role in metabolism other than its role in clotting, as had been postulated. The fact that the direct effects of plasminogen deficiency were ameliorated by fibrinogen deficiency showed that plasminogen probably had no other role.
4. <http://udel.edu/~mcdonald/oldmousetrap.html>. Professor McDonald has recently designed a new series of traps that can be seen at <http://udel.edu/~mcdonald/mousetrap.html>. I have examined them and have concluded that they involve his directing intelligence to the same degree.
5. M. J. Behe, "A Mousetrap Defended: Response to Critics." <www.crsc.org>
6. <http://biocrs.biomed.brown.edu/Darwin/DI/Mousetrap.html>

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The Cambrian Information Explosion

Evidence for Intelligent Design

Stephen C. Meyer

INTRODUCTION

In his book *The Philosophy of Biology,* Elliott Sober (2000) notes that many evolutionary biologists regard the design hypothesis as inherently untestable and, therefore, unscientific in principle simply because it no longer commands scientific assent. He notes that while logically unbeatable versions of the design hypothesis have been formulated (involving, for example, a "trickster God" who creates a world that appears to be undesigned), design hypotheses in general need not assume an untestable character. A design hypothesis could, he argues, be formulated as a fully scientific "inference to the best explanation." He notes that scientists often evaluate the explanatory power of a "hypothesis by testing it against one or more competing hypotheses" (44). Thus, he argues that William Paley's design hypothesis was manifestly testable but was rejected precisely because it could not explain the relevant evidence of contemporary biology as well as the fully naturalistic theory of Charles Darwin. Sober then casts his lot with modern neo-Darwinism on evidential grounds. But the possibility remains, he argues, "that there is some other version of the design hypothesis that both disagrees with the hypothesis of evolution and also is a more likely explanation of what we observe. No one, to my knowledge, has developed such a version of the design hypothesis. But this does not mean that no one ever will" (46).

In recent essays (Meyer 1998, 2003), I have advanced a design hypothesis of the kind that Sober acknowledges as a scientific possibility. Specifically, I have argued that the hypothesis of Intelligent Design can be successfully formulated as "an inference to the best explanation" for the origin of the information necessary to produce the first life. Such a design hypothesis stands, not as a competitor to *biological* evolutionary theory (i.e., neo-Darwinism), but instead as a competitor to *chemical* evolutionary theories of how life first arose from nonliving chemicals.