

## **The Bacterial Flagellum: A Response to Ursula Goodenough**

This article is a response to Ursula Goodenough, author and senior scientist at Washington University, St. Louis, who responded to an article I wrote critiquing Matt Young's scenario for how specified complex information can evolve naturally. The entire exchange has occurred on the science and religion forum Metanexus (www.metanexus.net), and the articles may be found at the following locations:

Matt Young's article "How to Evolve Specified Complexity by Natural Means":  
[http://www.metanexus.net/archives/message\\_fs.asp?list=views&listtype=Magazine&action=sp\\_simple\\_archive\\_&page=1&ARCHIVEID=5349](http://www.metanexus.net/archives/message_fs.asp?list=views&listtype=Magazine&action=sp_simple_archive_&page=1&ARCHIVEID=5349)

John Bracht's response to Matt Young, "Knotty Pine and Corroding Coins":  
[http://www.metanexus.net/archives/message\\_fs.asp?list=views&listtype=Magazine&action=sp\\_simple\\_archive\\_&page=1&ARCHIVEID=6385](http://www.metanexus.net/archives/message_fs.asp?list=views&listtype=Magazine&action=sp_simple_archive_&page=1&ARCHIVEID=6385)

Ursula Goodenough's response to Bracht, "Of Flagella and Outboard Motors":  
[http://www.metanexus.net/archives/message\\_fs.asp?list=views&listtype=Magazine&action=sp\\_simple\\_archive\\_&page=1&ARCHIVEID=7143](http://www.metanexus.net/archives/message_fs.asp?list=views&listtype=Magazine&action=sp_simple_archive_&page=1&ARCHIVEID=7143)

The current essay is a response to the last article by Goodenough, and has also appeared on Metanexus:

Part 1:  
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Part 2:  
[http://www.metanexus.net/archives/message\\_fs.asp?list=views&listtype=Magazine&action=sp\\_simple\\_archive\\_&page=1&ARCHIVEID=7790](http://www.metanexus.net/archives/message_fs.asp?list=views&listtype=Magazine&action=sp_simple_archive_&page=1&ARCHIVEID=7790)

Both parts were initially a single article, which has been slightly revised and may be found in its entirety here.

I appreciate Ursula Goodenough taking the time to write a lengthy response to my article “Knotty Pine and Corroding Coins,” which was itself a response to Matt Young. Goodenough raises some important issues, and has spurred my thinking and research deeper into the scientific literature of the bacterial flagellum, for which I am grateful. In her article, Goodenough begins by reprimanding me for not referencing the primary technical literature on the bacterial flagellum. While my original essay was primarily intended for a lay audience and thus the technical content was minimized, I do appreciate the need for referencing the scientific literature. Interestingly, Goodenough doesn’t claim that any of my technical comments about the bacterial flagellum are factually incorrect or that either Behe or Dembski’s technical details about the flagellum are wrong. Rather, she chooses to focus on the origin of the bacterial flagellum—for which she provides speculation and ideas but (curiously) no references to the technical literature (or any literature whatsoever) to support these hypotheses. As I will argue, the evidence actually calls into question the evolutionary history she proposes.

To give a quick overview of the flagellum, it is fitting to quote one of the experts Goodenough chides me for not citing in my original article, David DeRosier:

More than actomyosin or tubulokinesin, the bacterial flagellum of *Salmonella typhimurium* is the analogue of a man-made mechanical system. Its heart is a 15 000 revolutions per minute, reversible rotary motor powered by the proton-motive gradient across the cell's inner membrane. Each revolution consumes about 1000 protons. A drive shaft, held by a bushing in the outer membrane, transmits torque across the cell's envelope. Attached to the drive shaft, a universal joint enables the motor to drive the propeller, even when the drive shaft and propeller are not co-linear. A short junction joins the propeller to the drive shaft. The propeller, a long left-handed corkscrew, converts torque to thrust. A cap sits at the cell distal end of the filament. By electron microscopy, the motor associated parts and the bushing are seen to be rings of subunits, whereas the drive shaft appears to be a helical assembly of subunits. About four dozen genes are needed to build the flagellum. Some are required for regulation of synthesis; some for export and assembly; some for the structure itself, and a few are of unknown function. Nineteen different proteins are known to be part of the flagellar structure; it is thought that there may be additional components.

[source: DeRosier D. Spinning Tails. *Curr Opin Struct Biol.* 1995 Apr;5(2):187-93]

The bottom line is that the flagellum consists of a tightly-integrated system in which scientists have discerned essential functions for each of the components. Knock-out experiments have shown that eliminating any of these flagellum proteins results in a nonfunctional machine. My argument (in my previous essay) was that this functional holism, the fact that each protein is required for function, poses a strong challenge to Darwinian explanation. All the different functional components would have to come together in a single step (whether the proteins spring up brand-new, are co-opted from

pre-existing systems, or arise from gene duplication), falling together at the same instant, to achieve selectable function and be preserved. Given what we know about the bacterial flagellum and the engineering constraints upon the system, this would appear to be a compelling argument. However, in an act she calls “blowing the whistle,” Goodenough writes the following:

The fact that the modern components of the modern bacterial flagellum are required for function—mutations in any component destroy function—says nothing about the original properties of the intermediates. A protein originally "added to" a primal flagellum (the precursor to protein #18 of the modern flagellum, let's say) does not need to be posited as having been necessary to primal flagellar function at all. Its mutation-conferred chance ability to associate with the primal flagellum would by definition be an "extra," since the primal flagellum had, by definition, some sort of a functionality on its own or we wouldn't call it a flagellum. All we need to posit is that when the original version of #18 added on, the primal flagellum worked better in some way, the creature was "fitter," natural selection operated, and the gene spread. Now enter a process abundantly documented in the biological literature but ignored in ID arguments with which I am familiar, namely, the process of co-evolution. If our protein #18 initially conferred its freebie advantage by binding to pre-existing protein #9, let's say, then what happens during co-evolution is that any mutations in protein #9 that permit #18 to bind better, or better confer its selectable advantage, are also going to spread, as will any mutations in #18 that permit #9 to better bind to it or help it in its function.

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This is the heart of her argument—that the flagellum began as a simple system which performed its function and gradually increased complexity by accruing additional “freebies” that were enfolded into the nascent structure. Indeed, this is the only reasonable way for the Darwinian mechanism to work, and as long as the story is kept vague and speculative, it seems reasonable. However, as a scientist I want to try to think clearly about this scenario and its implications. Notice that Goodenough posits a “primal flagellum” that lacked protein #18. It appears, then, that she is assuming that this primal system lacked one of the components (protein #18) and was still functional. But on what basis does she assume this precursor would be functional? The evidence from DeRosier suggests that each protein plays a crucial role in the system, and conceptually we can see why deleting any one of these proteins will result in a nonfunctional flagellum. Regardless of this evidence, Goodenough simply posits an intermediate flagellum that lacks one or more of these essential proteins but still performs its function. Indeed, Goodenough insists that “the primal flagellum had, by definition, some sort of a functionality on its own or we wouldn't call it a flagellum.” The circularity here is extreme: obviously, if we assume that only a functional system would be labeled as a

flagellum, then of course whatever we call a flagellum will be functional; the question, then, is this: would we be able to call the intermediate structures “flagella”? By assuming the answer to this question is yes, Goodenough simply begs the question. But this tells us precisely nothing about the reasonableness of Goodenough’s assertion that her intermediates would, in fact, be functional. Indeed, in light of the results of knock-out experiments, it is reasonable to argue that Goodenough’s primal flagellum would not be functional—because it would lack an essential protein.

When we begin to probe Goodenough’s scenario further, we run into more problems. She insists that the primal flagellum was, indeed, functional, which suggests that the primal flagellum somehow was already managing to perform all the required functions for flagellar motility, without the contribution of protein #18 (which we assume was lacking to start with). This means that somehow *the primal flagellum was compensating for the lack of protein #18*. This leads to an interesting problem: if the flagellum was already performing the function that later would be done by protein #18, what is the selective advantage of having protein #18 added to the system? It appears not to be adding anything really new beyond what the system already is doing. There shouldn’t be any selection pressure for preserving it. We run into the conundrum that if protein #18 performs an essential function the machine will be functionless without it, contra Goodenough’s analysis. And if Goodenough is right, and the primal flagellum functions fine without protein #18, there is no advantage to adding it to the system; it’s not doing any function that the system doesn’t already perform.

But of course, Goodenough would argue that merely by binding to the pre-existing primal flagellum, protein #18 is providing selectable advantage. But why should we think that mere “binding” is automatically going to provide an advantage? Indeed, there are good reasons to think that generic binding of proteins to functional complexes is likely to gum up the original function and possibly destroy the machine. Imagine a protein that gets stuck in the proton-translocation pores of the flagellum or gets jammed between the motor proteins, preventing torque generation; certainly function would be reduced or destroyed. But let’s assume that this binding interference doesn’t happen, and that protein #18 successfully sticks itself somewhere on the functional complex without interfering with any other functions. What could it possibly contribute to the system to make it selectable advantageous? This is where the vagueness of Goodenough’s story makes it hard to critique. Goodenough never tells us what function protein #18 will eventually perform, what functions it might have originally had, what the primal flagellum did to compensate for lacking protein #18 originally, and how protein #18 became enfolded into the pre-existing system without disrupting it.

However, it is easy to see that adding a new protein to something like the bacterial flagellum is not trivial. Not only must the protein bind the complex, but its binding must be very specific to the types of proteins it must interact with. In the absence of any details from Goodenough, let’s assume that protein #18 is a precursor for FlgE, the hook protein which joins the driveshaft and propeller and will ultimately function as a universal joint for the flagellum. Let’s think about what that proto-FlgE protein will need. It’s got to be able to form a polymeric structure with itself, so it’s got to have the ability to recognize and bind to itself to form a flexible, stable structure. But it also has to be able to bind to the two flagellum components it links together: the flagellin propeller and the driveshaft. Thus, it needs at least 3 binding sites with the right affinities for the right proteins. The

hook provides the ability to transmit force from the driveshaft to the propeller even when they are not coaxial; this imposes another constraint on protein #18, that it must exhibit a certain amount of *collective* conformational flexibility (remember, the hook composed of multiple flgE proteins). Furthermore, the proteins making up the driveshaft and propeller will have to change, since initially they bind to each other and must be converted to bind to the hook (which will serve as a linker between the two). It is certain that merely “binding” of the pre-flgE protein to the flagellar complex is not enough to confer selectable advantage—there are a host of engineering constraints governing how the protein and overall system interact, and it is the generation of these precise interactions that is the challenge to the Darwinian paradigm.

Part of the disagreement between Goodenough and myself stems, I think, from a divergence of views of what, exactly, biological functionality entails. My own studies of biology have amply demonstrated that biological functionality is turning out to be much more highly specified and precise than we had originally envisioned. Goodenough appears to envision biological systems exhibiting a sort of “squishy,” loosely defined collective function, where an extra protein may simply be tossed in to provide a “freebie” function. This contrasts with my own view, which is that biology is really a science of engineering, where the constraints for biofunctionality are extreme—to the point that nearly every molecular interaction is remarkably precise and tightly controlled. Molecular biology is much like a jigsaw puzzle where each piece must be specifically shaped to fit with the other pieces around it. The pieces (proteins) are shaped such as to fit together in one, and only one, way—and if they fail to do so, disease and death usually result. For examples, just look at sickle-cell anemia (where hemoglobin proteins with incorrect affinities for each other can cause disease) or Alzheimer’s disease (where misfolded protein plaques cause damage to nerve cells) or prion diseases (caused entirely by misfolded, misshapen proteins); each of these diseases involves proteins “fitting together” in the wrong way. Enzymes function by precise interactions based on their 3-dimensional conformation relative to their substrate molecules; molecular biologists speak of enzymes as “locks” with corresponding substrate “keys,” describing the highly precise match between the two. More recent work has begun to uncover higher-order enzyme complexes wherein enzymes that act sequentially in a pathway are physically grouped together and can hand off their substrates to one another. In a similar but more precise way, biomolecular machines consist of tightly-integrated protein complexes that exhibit complementary interlocking surfaces which collectively build driveshafts, bearings, and motors. Not only do these proteins have precise and specific affinities for each other, their expression patterns are tightly controlled so that the cell doesn’t make the wrong protein at the wrong time. The flagellum has a built-in quality-control system that terminates flagellar protein production if any step of the assembly process goes awry. In light of this emerging biological reality, it is no longer reasonable to speak of just throwing a new protein into a system to produce selectable advantage; rather, biochemical explanations must account for the production of specific complementary surfaces, the keys that turn biochemical locks and bring together the jigsaw puzzles that constitute the cell’s machinery. Evolutionary explanations must describe how a new protein integrates into an old system in such a way as to allow continued functionality overall (often, both the incoming protein and the pre-existing system must be extensively

modified to fit together in a coordinated way), and enhance functionality of the entire system in such a way as to provide selective advantage.

One consequence of this divergence of views is that Goodenough thinks I am being disingenuous in using the current engineering constraints on the bacterial flagellum to argue that it would be impossible to evolve the system gradually. This explains her language about “blowing the whistle” and her statement that I am “constructing a house of cards.” She merely insists that the flagellum *did* have functional precursors and gradually enfolded “freebie” proteins along the way to become what we see today. Part of the weakness of this theory is that if it is true, we can never know it from current evidence, since it explicitly argues that we cannot deduce the past state of a system from its current state. This allows for endless, uninhibited speculation that is based entirely on a conviction that natural selection can do all the design work needed. It’s a story that is simply taken as true; it is untestable, and thus, unfalsifiable. But why should we believe such a just-so story? I, for one, would be much happier if Goodenough could explain what the proteins were in the primal flagellum, what functions each protein performed, and how new proteins were enfolded into the system. Merely asserting that this happened is simply not sufficient to scientifically explain a phenomenon, especially in light of the fact that we have a conceptual and experimental framework that suggests strongly that Goodenough’s scenario *wouldn’t* work.

It is also worthwhile to ponder the following conundrum: assuming Goodenough’s story is true, and biological function can arise from loose conglomerations of proteins, how and why would such loose conglomerations of proteins evolve into the tightly specified, integrated systems we see today? If the loose conglomeration will do the job, what’s the benefit of having a tightly specified version (especially considering the vast improbabilities associated with getting all the interlocking proteins just right in a chance-driven step)? This points to a real problem: even if the precursor system was a loose and non-specific conglomeration of proteins, the present-day system is, in fact, not this way. This means that at some point, a transition had to occur wherein the modern-day engineering constraints upon the bacterial flagellum came into play. Whatever that transition point was, all its novel and precise protein interactions had to come into existence *all at once*—since we know, experimentally, that the modern flagellum requires all its proteins for function, and this was the transition which produced the modern flagellum. In other words, Goodenough’s model entails a crystallization from loose conglomeration to tightly engineered modern flagellum which had to occur in a single, coordinated step (again, because we know that all the proteins in a modern flagellum are required for function).

And what sort of selective pressure would drive such a transition? Perhaps the Darwinian explanation would suggest that a loose conglomeration of proteins is not as efficient as a more tightly integrated one; hence the latter will have a selective advantage. However, I can utilize the same imprecision exploited by the Darwinist and insist that this loose conglomeration was actually highly efficient (after all, the evidence says precisely nothing about this loose conglomeration of proteins either way, so I am just as free to speculate as the next person). Perhaps this is the best way to see the truly unscientific nature of this entire line of argumentation.

The bottom line is, if a slow, gradual evolutionary process is truly how the bacterial flagellum came into existence, then there was a chain of intermediates that led to the

modern form. We should, in theory, be able to trace that chain backward from the modern system, if not experimentally via knock-outs, at least theoretically by postulating what intermediates looked like and how they were interconverted. Rather than provide this sort of scientific rigor, Goodenough proposes a remarkably vague story that actually contradicts the available evidence about the bacterial flagellum; this looks suspiciously like a deliberate retreat into vagueness in the face of evidence that threatens one's favorite hypothesis. Indeed, if I were to presume as much, I might dare to suggest that it is Goodenough who is trying to construct a house of cards without any evidence to support it.

Ironically, in this case, it is Darwinian theory that is functioning as an "evolution-of-the-gaps" theory. Faced with the remarkable inter-dependent complexity of the bacterial flagellum and no apparent way to build functional intermediates, the committed scientist just plugs in unknown, unknowable precursors that are not only unsupported by the evidence, but are actually antithetical to what that evidence does say. When the rubber hits the road, it is little more than a science-stopping argument from ignorance in the face of a real, ontological gap in the history of life. The origin of the bacterial flagellum rips a hole in our explanatory framework (Darwinian theory) that is not going away any time soon.

I want to address a system that Goodenough briefly mentioned as a possible functional precursor to the bacterial flagellum: the ATPase:

"And likewise, the very first flagellum might have been nothing more than a rotating ATPase (found throughout the biological kingdom) with some fibrous protein (independently a cell-wall component or some such) attached to it. Sounds "complete" enough to move you through the water a bit more than if you didn't have one, and adaptive enough to be a substrate for natural selection."

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As interesting as this story is, it fails to account for the evolution of the flagellum as soon as we begin considering the details of the ATPase and cell structure and function. For brevity, I will quickly list a few reasons why the ATPase story would not work:

1. The ATPase of bacteria faces *inward* from the cell membrane, so the rotary portion would never be exposed to the external environment as would be required for any bacterial flagellum precursor.
2. The ATPase requires several chaperone proteins for its proper assembly, so it could never be assembled facing outward—the requisite chaperones (which are located in the cytoplasm) would not be able to assist the assembly process. (for details, see the Ackerman reference)
3. The proton gradient that drives the ATPase is the wrong way for a "backward" ATPase facing the environment—so even if the ATPase were reversed (somehow overcoming the assembly problem), it would not be able to rotate; hence it would be nonfunctional.

4. The ATPase performs the vital function of synthesizing ATP for the cell. This synthesis of ATP occurs at the rotation portion of the enzyme, so if the ATPase were inverted (facing outward from the cell), the ATP it synthesized would be lost to the external environment. Without this source of energy, the cell would die. If some ATPases were left unmodified, we may reasonably ask about the origin of the added genetic regulatory systems that choose to specifically invert only some ATPases and not others.
5. A straight propeller is not enough to confer motility. Researchers have found that mutations in the flagellum filament that result in a straightening of the filament eliminate motility (see the DeRosier reference). Thus, an ATPase with a straight piece of fiber would be useless as a motion device.
6. How would an ATPase be converted into a flagellum? The problems are immense, starting with the fact that the ATPase has only one proton pore in the middle, with the rotating portion around the pore. The flagellum is the opposite of this, with the proton pores around the edges and the rotary rings located in the center. Furthermore, the flagellum's basal body (including motor, stator rings, driveshaft, etc) is about five times larger than the entire ATPase, and incorporates a protein-pump that allows the flagellum to be built from the inside out. This difference in size and complexity is reflected in the number of proteins in each system; the flagellum has around 20 different structural proteins, while the ATPase utilizes only 8. Adding a flagellum-type pump functionality to the ATPase would be a massive re-engineering problem in which the original structure would have to be entirely disrupted and many novel proteins inserted in precisely the right positions and orientations to perform the new function. There is no way to gradually add the ability to secrete proteins to the ATPase system—like the bacterial flagellum overall, the pump function requires certain tightly interacting proteins that must all function together.

For the sake of argument, let's grant that the ATPase and attached fibrous protein were able to rotate in the environment outside the cell, and consider the ATPase/fiber system in relation to the environment the bacteria actually encounters. Because of the small size of bacterial cells, individual water molecules are actually able to knock them around in a watery environment—an effect known as Brownian motion. If you have ever seen bacteria in a watery media under a microscope, you know exactly what I am talking about: they vibrate and bounce around quite chaotically unless they are affixed somehow to a substrate. Certainly, free-swimming bacteria are not affixed to anything, and hence they are subjected to the randomizing effects of Brownian motion. As an analogy to the ATPase “flagellum,” imagine being on a small boat, swept downstream in a raging torrent of a river. To help you navigate, the boat has a small outboard motor equipped with a propeller that rotates 3 times per minute. Would this work to get you anywhere? No—you would be carried downstream at the whim of the river. The environmental conditions are such that a basic level of functionality (i.e., speed and power of the motor) is required before the motor does you any good at all. The same is true for the bacterial flagellum: in their chaotic environment, a mere fiber projecting from an ATPase molecule on a bacterial cell is not going to be enough to meet even the basic requirements for motility. The flagellum is a massive engine (as observed before, the basal body alone is around five times larger than the entire ATPase) which rotates at 15,000rpm and has an

absolutely enormous (relative to the bacterial cell) propeller to counter the strong destabilizing effects of Brownian motion (remember, the flagellum can be ten times the length of the bacterial cell). It's a major powerhouse to propel the cell through its chaotic environment. Anything significantly less powerful is going to be utterly ineffective in providing selective advantage.

Finally, I want to address the issue of gene duplication which Goodenough suggests as a mechanism for evolutionary innovation. My analysis here will counter both gene duplication and cooption arguments, and it rests on the fact that generating novel biofunctionality, when properly understood, is not an issue merely of generating new proteins. Rather, it is a matter of generating *new interfaces between proteins, and generating new regulatory controls on those interfaces*. In other words, biological evolution has to explain how the jigsaw puzzle pieces came to fit together. Sure, the jigsaw pieces were maybe originally copied from each other, but they then had to have unique knobs, bumps, and indentations added to give them the ability to interact in unique and new ways. Novel biological functions require novel protein interactions, and a system like the bacterial flagellum contains many highly specific interactions. It is the origin of these interactions, not the origin of the proteins themselves, that is at issue. The protein starting material can come from anywhere—duplicated genes, genes originally serving another function, whatever. The point is that these genes are already serving a function elsewhere in the cell. Gene duplication only gives you a protein which does the same thing as another protein—the real challenge is to explain how and why that gene acquires genuinely new functions. So perhaps there were 50 gene duplications prior to the evolution of the flagellum to provide the raw material for eventually generating 50 flagellar genes, but the real challenge is to explain how these genes came to function together in the novel flagellar system. In fact, it is even possible that these 50 extra genes (freshly duplicated) mutated in such ways that they formed a loose conglomeration of proteins at some point, as Goodenough argues. But somehow, at some point, they had to acquire the highly specific, tightly integrated functional characteristics they exhibit today. And again, since we know that knocking out any protein from the flagellum destroys function, they must all have made the “leap” to tight, integrated function *at precisely the same time* to come together into the modern flagellum. The reasoning is simple: imagine that all but one protein in the proto-system made the transition from loose conglomeration to tight integration. The resulting system would be equivalent to our single-gene knock out experiment (it would lack one tightly-integrated protein), which we know is functionless. Certainly, if this is true of any one protein in the system, it will be true of subsets of proteins in the system. Therefore, we can safely state that *at some point in its evolutionary history, the primal flagellum underwent enormous, coordinated mutations that brought together the entire system into a tightly integrated, functional complex*. It doesn't matter whether the proteins that underwent this transition came from gene duplications, cooptions, non-coding sequences, whatever. It doesn't matter if, in the process, the pre-flagellar proteins underwent stages in which they formed some poorly-defined sort of loose conglomerate (although we don't have either conceptual or experimental evidence to support this idea). The problem is that the proteins which are to become the flagellum are coming from systems that are distinctly non-flagellar in nature (after all, we are discussing the origin of that very system) and being co-modified from their original molecular interactions into an entirely new set of molecular interactions.

Old interfaces and binding sites must be removed and new ones must be created. But given the sheer number of flagellar proteins that must co-evolve and the fact that the entire change must happen in one step, co-generating all the proteins required for flagellar function (again, this is true *at some point in the flagellum's evolutionary past even if there were earlier steps that were not so tightly constrained*), the Darwinian explanation is really no different from appealing to a miracle.

It's been said that science is the orderly arrangement of what, at the moment, seem to be facts. Ursula Goodenough goes this one better. At her hands evolutionary biology becomes a systematic rationalization of what, for all we can tell, is a long string of miracles.

**John Bracht**  
**September 22, 2002**

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