

# Redundant Complexity:

## A Critical Analysis of Intelligent Design in Biochemistry.

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### *Abstract.*

Biological systems exhibit complexity at all levels of organization. It has recently been argued by Michael Behe that at the biochemical level a type of complexity exists -- *irreducible complexity* -- that cannot possibly have arisen as the result of natural, evolutionary processes, and must instead be the product of (supernatural) *intelligent design*. Recent work on self-organizing chemical reactions calls into question Behe's analysis of the origins of biochemical complexity. His central interpretative metaphor for biochemical complexity, that of the well-designed mousetrap that ceases to function if critical parts are absent, is undermined by the observation that typical biochemical systems exhibit considerable redundancy and overlap of function. Real biochemical systems, we argue, manifest *redundant complexity* - a characteristic result of evolutionary processes. (.We would like to thank George Gale for helpful comments, as well as the anonymous referees for *Philosophy of Science*.)

### **1. Introduction.**

The biological hierarchy of organization runs from macromolecules, to intracellular structures, cells, tissues, organs, organisms, populations, ecosystems and ultimately to the biosphere itself. Considerable complexity is observed at all levels, and there are complex relationships between levels in the hierarchy. Evolutionary biology is currently our best theory of the nature of complex biological systems.

Recently, Michael Behe (1996) has argued that there is a biochemical objection to evolutionary biology. According to Behe, biochemists have uncovered a kind of complexity -- *irreducible*

*complexity* -- that cannot possibly have its origins in natural, evolutionary processes. This claim, widely reported, has now become a centerpiece of the new, *academic creationism* -- Phillip Johnson, for example, bases part of his popularization of *intelligent design biology* on Behe's "discovery" (1997, pp. 76-77). We argue that Behe is profoundly mistaken about the nature of biochemical complexity. But while a critical examination of Behe's arguments and claims may be important, especially in view of the way so-called "scientific" creationists are starting to use them in discussions about public education, the true philosophical value of such an analysis lies in the light it sheds on the nature of biochemical complexity itself.

But does irreducible complexity represent a phenomenon beyond the domain of evolutionary biology? In our argument, we do not claim to fill in the current gaps in our knowledge where Behe and others wish to insert an intelligent designer. Rather, we try to show how it is *possible* to fill in the gaps in a naturalistic, evolutionary fashion. As to how the current gaps in our biochemical knowledge can be filled in, only the course of future research will tell.

After presenting Behe's central argument, we argue first, that *self-organizing* chemical reactions that satisfy Behe's criteria for irreducible complexity are neither mysterious nor naturalistically inexplicable. Such reactions arise outside biochemistry in the context of a class of reactions in organic chemistry, where the chemical mechanisms and patterns of dynamical activity are well-understood. Second, we show that processes exhibiting similar patterns of dynamical activity are found at various levels of the biological hierarchy of organization, where they have been co-opted in the course of evolutionary time to serve a variety of adaptive ends. Thirdly, we argue that evolved biochemical systems actually exhibit *redundant complexity* -- a kind of complexity that calls into question the validity and applicability of Behe's central interpretative metaphor -- that of the *well-designed, minimalist mousetrap*. But first, what are mousetraps doing in a debate about the evolution of biochemical complexity?

## **2. Irreducible Biochemical Complexity: Behe's Mousetrap.**

Behe's central thesis is that processes of the kind invoked by evolutionary biology cannot explain the origin of irreducibly complex structures and processes of the kind observed in the biochemistry of extant organisms. Behe tells us that:

By *irreducibly complex* I mean a single system composed of several well-matched, interacting parts that contribute to the basic function, wherein the removal of any one of the parts causes the system to effectively cease functioning. An irreducibly complex system cannot be produced directly (that is, by continuously improving the initial function, which continues to work by the same mechanism) by slight, successive modifications of a precursor system, because any precursor to an irreducibly complex system that is missing a part is by definition nonfunctional. [1996, p. 39]

For Behe the implication is clear: if these systems could not evolve, they must have been intelligently designed. Leaving aside the false dilemma, what exactly defines an irreducibly complex system?

According to Behe, irreducibly complex systems have two essential characteristics [1996, pp. 42-43]. First, the system must be composed of several key components, all of which contribute to the function achieved by the system *as a whole*. Second, the key components must be essential for the achievement of the system's function(s). Behe employs an analogy with well-designed, minimalist mousetraps. Such a mousetrap has several components, all of which contribute to its ability to catch mice, and all of which are needed for this purpose. A precursor "trap" that lacked one of the components -- the spring, the trigger or the platform, perhaps -- could not trap mice anyhow, and lacking even minimal function, could not be improved through step-wise adaptive evolution into a functioning trap.

Behe's central argument boils down to the claim that systems satisfying certain conditions -- systems consisting of several key components, all contributing to the function(s) achieved by the system as a whole, and all essential to the achievement of those systemic functions -- cannot have their origins in unintentional, natural processes. Such systems require a supernatural, intelligent designer. Hence, if we can formulate plausible naturalistic explanations (devoid of appeals to designers of any kind), for some systems satisfying Behe's criteria, then we have reason to question the general validity of his claims.

### **3. Self-Organization and Irreducible Complexity.**

Biochemical complexity is not just manifested in the complexity of biomolecules themselves. Rather, there is a complex system to life -- an integrated, interactive system whose properties as a whole exceed those of the individual parts. The problem of biochemical complexity thus has a dynamical component. Understanding the nature of this dynamical component, with its consequences for the emergence of system-level properties, will enable us to understand how to explain the origins of *irreducible complexity*.

One of the aims of *complexity theory* is to provide rigorous explanations of the emergence of *self-organization* -- the spontaneous formation of ordered, interactive complex systems, structures and processes. Systems satisfying Behe's criteria for irreducible complexity, are also systems in the domain of complexity theory. Behe recognizes the relevance of the concept of self-organization referred to in complexity theory, and the threat it poses for the complexity problem he believes evolutionary biology must confront.

Taking the abstract, mathematical work of Stuart Kauffman [1993] as the exemplification of complexity theory, Behe criticizes complexity theorists for not paying attention to the details of biochemical reactions. Kauffman's mathematical models of self-organizing processes often do not mention specific biochemical details and there is thus ". . .the tendency to get further and further away from real chemistry and to get trapped in the mental world of mathematics" [1996, p. 156]. God and not the Devil, Behe contends, is in the irreducible complexity of the details. He is thus obliged to dismiss complexity theory as an irrelevant, "fact free" science.

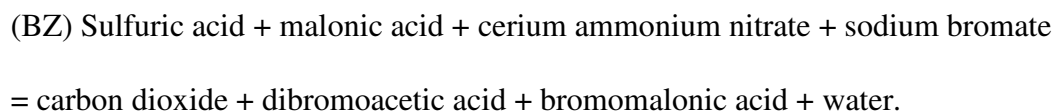
Following Behe's advice, let's look at some chemical details. In what follows, we will consider a reaction from organic chemistry -- the Belousov-Zhabotinsky (BZ) reaction -- and its direct

significance for our understanding of the origins of Behe-type irreducible complexity. Our analysis will also show how principles from complexity theory can be used to illuminate real chemical and biological systems, thereby closing some gaps in our knowledge, and showing how, in principle, other such gaps might be closed without the insertion of supernatural, intelligent designers.

The BZ reaction was first observed in the fifties, when B.P. Belousov was attempting to simulate the Krebs (citric acid) cycle *in vitro* (Winfrey 1984). The Krebs cycle involves a sequence of biochemical reactions fundamental to the metabolism of aerobic organisms. The Krebs cycle has several component reactions, and is called a cycle because the reaction sequence goes round in a loop (it is in fact a biochemical oscillator). The *complete* disruption of a key reaction in the sequence would disrupt the entire cycle. The Krebs cycle qualifies as an exemplar of Behe-type irreducible biochemical complexity.

When Belousov ran his reaction, he got a surprise. He found that his test tube contained a chemical oscillator. Subsequent work has shown that this reaction too, consists of a reaction sequence that forms a loop. This reaction, like the Krebs cycle it models, exhibits irreducible chemical complexity. What Belousov had discovered was in fact a self-organizing chemical reaction. In the decades that followed, the chemical mechanism has been elucidated, other BZ-type systems have been discovered, including biological systems, and a mathematical model describing the BZ-type dynamics has been elucidated.

It is instructive, then to consider some of the details concerning our knowledge of BZ-type systems. There are several distinct recipes for BZ-type reactions (Tyson 1994), but a commonly used one has the following global stoichiometric equation (Babloyantz 1986, p. 156):



Like the Krebs cycle, this reaction involves the oxidization of an organic substrate.

When the reaction is run in a test tube, oscillations in color are observed as the chemical system cycles through its component reaction pathways.

In the first group of reactions, bromide ions, bromate and malonic acid are used in a slow reaction to produce bromomalonic acid and water. Bromous acid is one of the reaction intermediates in this pathway. Since the cerium present is in the cerous state, the reaction medium remains colorless for this phase of the cycle. As the reaction proceeds the concentration of bromide ions decreases to a critical threshold, at which point enough bromous acid is present to open a more efficient pathway for the production of bromomalonic acid.

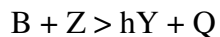
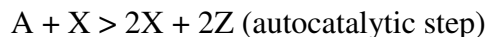
Here, in a fast reaction, bromate, malonic acid, bromous acid (a reaction intermediate of the first pathway) and cerous ions produce ceric ions, bromomalonic acid and water. The reaction medium turns yellow as cerium enters the ceric state. This pathway also contains an autocatalytic step in that one mole of bromous acid produces two moles of bromous acid. As the cerous ions

are consumed and ceric ions accumulate, a critical threshold is achieved at which time a third pathway opens which consumes bromomalonic acid, malonic acid and ceric ions to produce carbon dioxide, bromide ions, and to regenerate cerous ions (thus turning the reaction medium clear), and setting the system up for the beginning of a new cycle. The system will oscillate in color until equilibrium is achieved -- in typical demonstrations, we have observed regular oscillations for more than thirty minutes. The frequency of oscillation is a function of temperature and initial concentration of constituents.

This reaction satisfies Behe's criteria for irreducible chemical complexity since in this reaction sequence there are many key components and pathways, and the characteristic behavior of the system is disrupted if key components (for example bromide ions or cerous ions) are absent at critical phases of the reaction (or somehow removed as they are produced). Irreducible complexity in a self-organizing system is something generated and sustained by natural mechanisms which can be elucidated without the aid of a designing *deus ex machina*. The patterns of behavior observed in this chemical system result solely from the operation of chemical laws and initial conditions.

In fact, our understanding of this curious reaction has been augmented by the employment of mathematical models from the field of nonlinear dynamics. Contrary to Behe's assertion, these mathematical models are not idle speculation, but are crucial to an understanding of real-world chemistry. For example, for some distances from equilibrium the system will behave as a chemical clock, its dynamics governed by a limit cycle attractor. For other distances from equilibrium, qualitatively new behaviors -- chaotic behaviors -- appear as the dynamics become governed by a strange attractor (the Simoyi-Roux attractor -- see Tyson 1994, Goodwin 1996). In such a dynamic regime, the chemical system can show emergent properties not explicable in terms of the properties of the components of the system.

The central dynamical model of the BZ reaction is the Oregonator. This model may be understood in terms of the following schema ( Babloyantz 1986 p. 174):



Here,  $A = \text{BrO}_3^-$ ,  $B = \text{bromomalonic acid}$ ,  $P = \text{HOBr}$ ,  $Q = \text{CO}_2$ ,  $X = \text{HBrO}_2$ ,  $Y = \text{Br}^-$ ,

$Z = \text{Ce}^{4+}$ , and  $h$  is a constant. The model (employed in commercially available software packages) can be expressed in terms of 3 ordinary differential equations that describe the complex dynamics of the reaction process (Tyson 1994, pp. 576-577).

The Oregonator, then, is a dynamical model in terms of which we can explain changes of chemical state over time. Being a dynamical model, and being concerned with the way a system changes its state over time, it can be used to describe behaviors of a given type -- let us call these behaviors *BZ-type behaviors*. BZ-type behaviors have been found in many systems, including biochemical and biological systems, though the substrates and products in these systems are very different from those in the BZ reaction.

To understand these other BZ-type behaviors, it is instructive to consider what happens when the BZ experiment is run with just a thin film of reactant in a petri dish. Waves of color change propagate through the medium. The color changes are waves of oxidation propagating through a reduced medium. The waves propagate because of diffusion of bromous acid ahead of the wave front and its autocatalytic production behind the wave front (Tyson 1994, p.579). While the waves may start propagating in the form of concentric rings (target patterns), disturbances in the medium (for example the introduction of an inhomogeneity by tapping the medium with a toothpick -- see Nicolis 1989 for high quality photographs) lead to the production of rotating spiral patterns.

In essence, the wave of oxidation rotates around the inhomogeneity (known as a pacemaker), and since spirals have a geometry that is just a bit more efficient at inducing the next wave of oxidation in the medium than concentric rings, in due course the spirals displace the concentric rings in the medium. This is a good example of an evolutionary chemical selection of an autocatalytically more efficient pattern. In addition to this, three-dimensional scroll waves have been observed to propagate in three dimensional gels of BZ medium (Babloyantz, 1986).

Once again, the Oregonator has been used to characterize these dynamical behaviors (Tyson, 1994, pp. 579-581). In its terms we have mathematical analyses of self-organization in time -- oscillations, and space -- propagating wave patterns. In other words, the spontaneous appearance of macroscopic ordered states of matter at scales orders of magnitude larger than those of fundamental reactions. Because the variables in the Oregonator can be re-interpreted in terms of other substrates and products, it is possible to extend the model to cover other systems of interest, including biochemical and biological systems.

For example, when food gets scarce, slime mold amoebae of the species *Dictyostelium discoideum* aggregate as the result of chemotaxis. In these circumstances we observe propagating target and spiral patterns as the amoebae, aggregating into slugs, signal to each other using waves of cyclic AMP that propagate by a diffusion-autocatalysis process analogous to that found in the BZ-waves, and with the same dynamical properties. The slugs are able to exploit the chemical dynamics for aggregation and differentiation, leading to dispersal, and subsequent replication. BZ-type dynamics are an integral part of their survival strategy.

The dynamical properties exhibited by the BZ reaction have also had applications to the onset of oscillations in the concentration of glycolytic intermediates in yeast cells as they oxidize sugar (Babloyantz 1984, p. 255). Winfree (1994) has shown how waves of contraction (electrical BZ-waves) propagate in heart tissue, with the switch from concentric rings to spiral waves (rotating round infarctions) being associated with the onset of ventricular fibrillation. There are many other examples, ranging from the role played by BZ-waves in the assembly of key intracellular

structures known as microtubules (Hess and Mikhailov 1994, Tabony 1994), to the organization of important aspects of developmental processes (Goodwin, 1996).

To summarize the argument so far, Behe's challenge is to explain the origins of irreducibly complex systems:

An irreducibly complex biological system, if there is such a thing, would be a powerful challenge to Darwinian evolution. Since natural selection can only choose systems that are already working, then if a biological system cannot be produced gradually, it would have to arise as an integrated unit, in one fell swoop, for natural selection to have anything to work on. [1996, p. 39]

The challenge is met by showing that the self-organizing dynamics of complex systems can lead to the spontaneous formation of the "integrated units" (e.g., the BZ-type reaction cycle) that Behe claims are naturalistically inexplicable.

In short, complexity theory predicts, and experiments confirm, that Behe's irreducibly complex systems, can result from the dynamical phenomenon of self-organization. Self-organization, resulting in what Kauffman terms "order for free," can be exploited with advantage by evolving biological systems. Behe's claim that complexity theory is nothing more than abstract, "fact-free" science, must itself be revised in the light of these well-known examples. Far from being a "fact free" science, there is a wealth of hard empirical data on self-organizing systems in chemistry and biology.

#### **4. Redundant Biological Complexity.**

We have just seen that the existence of systems satisfying Behe's criteria for irreducible complexity do not constitute a principled stumbling block to a naturalistic understanding of biochemical complexity. But how might biochemical complexity have evolved? There is reason to believe that the evolutionary process of *exaptation* (Gould and Vrba 1982) could play an important role in the evolution of biochemical complexity. Exaptation is the process whereby pre-existing structures and pathways are co-opted to serve new functions. In this process, initial rounds of selection are for novel function and efficiency. In later rounds of selection, there will be selection for regulation of function. One of the key concepts here is that of *gene duplication*, a process whereby a gene is doubled in a genotype. As a result of this process, one gene can continue the old function, while the duplicate is freed up to be co-opted to serve novel ends. It is also an important source of the *redundant complexity* observed in biochemical systems.

Redundant complexity is embodied in the discovery that biochemical processes frequently do not involve simple, linear sequences of reactions, with function destroyed by the absence of a given component in the sequence. Instead, they are the product of a large number of overlapping, slightly different and redundant processes. This feature of these processes turns out to lie at the heart of the stability that these processes manifest in the face of perturbations that ought to catastrophically disrupt systems conceptualized from the standpoint of Behe's central

interpretative metaphor of the well-designed, minimalist mousetrap -- the absence of any component of which should render the system functionless.

We argue here that the *redundant complexity* observed in real biochemical systems, processes and structures, strongly supports the conclusion that the mousetrap metaphor is a completely inappropriate tool with which to conceptualize biochemical complexity. This element of redundancy is absent in economically designed mousetraps, where all parts are essential to function. We will illuminate the concept of redundant complexity with examples from biochemical, genetic, molecular and genomic organization.

## **5. Biochemical Pathway Complexity.**

Any cursory examination of biochemical pathways shows that organisms are often not dependent on simple, linear -- hence catastrophically interruptible -- sets of reactions to produce a needed product. This can be seen in the depiction of some of the more prominent interlocking biochemical pathways. If we examine the central catabolic pathway of glycolysis, it looks like the product of one reaction is required as the substrate for the next reaction in the sequence. Thinking with the aid of a "glycolysis mousetrap model," one might expect that removing one component, either enzyme or product, would shut down the pathway and prevent the continual production of energy. In fact almost every step in this pathway is redundantly complex. As an example let us look at the first step, the production of glucose-6-phosphate from glucose, catalyzed by hexokinase.

The phosphorylation of glucose is the first step in glycolysis. Not only does hexokinase, the enzyme that catalyzes this reaction, activate the relatively stable glucose (Bennett and Steitz 1978), but it is a multipurpose enzyme that, in part, controls the rate of the first part of the glycolytic pathway by controlling the "decision" of whether to direct glucose to the building up of more complex molecules, anabolism, or harvesting the energy stored in glucose, catabolism. This decision is dependent only on the concentration of the substrates, products and various components of the pathway (Voet and Voet 1995).

One might assume, therefore, that this case is a good exemplar of the relevant features of Behe's mousetrap. Remove the enzyme and the reaction should stop. But this intuition rests only on a superficial characterization of this step in the pathway. Looking at the fine details -- where the Devil lurks -- reveals an unexpected complexity to what appears to be a simple, straightforward chemical situation.

In typical vertebrate tissue, redundant complexity is manifested in the existence of several different isoforms -- variants -- of hexokinase, and all are present, as a result of gene duplication, in varying proportions, in different tissues. Removal of a given isoform does not disrupt glycolysis, though it may have an effect on the efficiency with which a function is achieved. Depending on whether the tissue requires rapid utilization of energy (muscles), or is involved in converting the glucose to a storage form, glycogen (in the liver), the isoform proportions differ for these specialized functions. One of the isoforms is inhibited by glucose-6-phosphate, the

product of its reaction, whereas another is not. There is redundant complexity here, in just the first step of the glycolytic pathway, a seemingly simple, straightforward step.

Each of the components of the rest of the glycolysis pathway manifests similar redundancies. Remove glucose and the enzymes can utilize numerous other hexose sugars to supply the next product. Knock out an enzyme and the "glycolysis mousetrap" should fail. But which enzyme? Knock out one enzyme isoform and the other isoforms in the tissue can take over its function. Maybe not quite as efficiently, but efficiency is something that can be improved by natural selection over evolutionary time.

If you could succeed in removing all the isoforms, there are alternative pathways that can supply the needed products, such as the pentose phosphate pathway (Martini and Ursini, 1996). The pentose phosphate pathway itself is primarily used to produce NADPH, an important carrier of chemical reduction potential. The first reaction in this pathway is catalyzed by glucose-6-phosphate dehydrogenase (*G6PD*). Population studies indicate that there are some 400 *G6PD* variants or isoforms (Beutler, 1992). An enzymopathy is known whereby *G6PD* is completely absent (Vulliamy, *et al.*, 1992). But redundancy exists here as well, and there are routes around this deficiency too.

It is a hallmark characteristic of evolved biochemical systems that there are typically multiple causal routes to a given functional end, and where one route fails, another can take over. The existence of multiple isoforms of a given enzyme are evolutionary legacies -- legacies by means of which one and the same enzyme can be co-opted to serve specialized functions in specialized tissues.

## 6. Genetic Pathways.

Failure of a simplistic mousetrap model can also be seen in the redundant complexity of another level of biological organization -- genetic pathways. Trees get their mechanical support from substances called *lignins* -- complex phenolic polymers. An example from the study of pine trees -- which might be used to make the platforms for real mousetraps -- supports our redundancy thesis. In pines, lignin is polymerized from two monomers, p-coumaryl alcohol and coniferyl alcohol, with this latter being predominant, amounting to some 90% of the polymer units. In the biosynthetic pathway that leads to lignin, cinnamyl alcohol dehydrogenase (*CAD*) converts coniferaldehyde to coniferyl alcohol, the primary monomer in pine lignin (Ralph, *et al.*, 1997, p. 235).

A loblolly pine mutant, homozygous for the *cad-n1* allele, has been identified and studied. *CAD* activity is about 1% of that in the wild type, resulting in the accumulation of high levels of coniferaldehyde, the *CAD* substrate. Thinking with the aid of a "lignin mousetrap model," one would naturally expect this mutant to be functionally inviable as a result of this seemingly catastrophic reduction in *CAD* levels. Instead researchers found these mutants are viable pines that incorporate a novel monomer --dihydroconiferyl alcohol -- into lignin composition. This monomer amounted to some 30% of the polymer units, as opposed to 3% in a normal pine.

Contrary to expectations based on prior experience with pines, and certainly contrary to those based on assumptions derived from the "lignin mousetrap," the mutant pines grow normally. This case certainly illustrates the role played by biological redundancy, and the consequent functional stability of a biological system in the face of quite dramatic genetic perturbation.

Genetic examples of *conservation of function* are also relevant to this discussion. If genes regulating development, for example, are components of "genetic mousetraps," then one would expect those systems to be unstable with respect to gene substitutions from other species, where the genes substituted for those deleted, serve different functional roles in their natural organismic homes.

Consider the chorion gene complex in the fruit fly, *Drosophila melanogaster*, and in the silkworm, *Bombyx mori*. The genomic organization of these two species are very divergent. The chorion genes in these two groups, coding for proteins used in eggshell construction that have no obvious structural similarity, are used to make very differently structured eggs, and are transcribed differently during egg production. Despite these differences, when a *B. mori* chorion gene fragment was used as a replacement for a deleted gene fragment in *D. melanogaster*, the genes were transcribed with the correct tissue and temporal expression patterns (Mitsialis and Kafatos, 1984).

These systems show stability in the face of quite radical perturbation at the genomic level. A naturalistic, evolutionary explanation simply holds that the chorion genes in moths and flies descend from a common ancestor gene. The genetic distance between flies and moths is similar to that seen between birds and mammals -- 300 million years since divergence from common ancestors (Mitsialis and Kafatos, 1984). The respective chorion genes are subject to adaptive modification in the time following divergence, but their ancient regulatory regions are nevertheless conserved. What we see here is redundant complexity, contrary to expectations based on intuitions derived from "genetic mousetraps."

## **7. Genetic Knockouts.**

Another way in which we can test the "genetic mousetrap model" of organisms is simply to remove specific sections of their genomes. This procedure has recently been applied to mammals. Researchers can now target a specific gene in mice and "knock it out" (Travis, 1992). Such knockout mice are valuable models for human diseases and gene function experiments. However, such mice do not always manifest the expected deficits (Cooke et al., 1997), even for seemingly crucial genes, and when this happens, they serve as examples of the type of redundant complexity we have been discussing.

One example concerns the gene *p53*, originally identified as a tumor suppression gene, but which has subsequently been found to be involved in a number of fundamental cell processes, such as affecting gene transcription, acting as control points in the cell cycle, initiating programmed cell death, apoptosis, DNA replication and DNA repair processes (Elledge and Lee, 1995).

Looking at this case from the standpoint of a "genetic mousetrap model," one would naturally predict that the removal of this gene, involved as it is in all of these vital processes, would lead to catastrophic collapse of the developmental process -- a bit like removing the spring, trigger or platform from Behe's mousetrap. Such is not the case, since *p53* knockouts in mice yield viable, fertile offspring, although they are susceptible to the early appearance of spontaneous tumors (Dowehower, et al., 1992). This suggests the following dilemma: either *p53* is not required for embryonic development or there are redundant ways in which the function of the missing component is compensated for (Elledge and Lee, 1995). The evidence at hand supports redundant complexity, since there are at least 400 proteins associated with the proper control of the cell cycle alone (Murray and Hunt, 1993), and it would appear that some of these other proteins pick up the slack created by the missing *p53*.

## **8. Genomics.**

The study of genome sequences -- albeit relatively new -- has revealed some startling findings about the complexity and organization of biological organisms. The genome of the yeast, *Saccharomyces cerevisiae*, contains many redundant sequences. Fifty three duplicated gene clusters, comprising 30% of the yeast genome, have been identified (Human Genome News, 1996, vol 8, pp. 5-7; Clayton, et al., 1997). Such findings concerning gene duplication lead to a question of fundamental importance for Behe's use of mousetrap metaphors. How few genes does it take to maintain a free living organism?. Experiments are currently underway to knock out all seemingly nonessential genes from the yeast genome.

If such experiments are successful, the resulting "minimal" organism will almost certainly not be able to survive except in a rigid set of conditions, and probably will not be able to regain equilibrium if the environment changes -- hence the importance of redundancy. But such a minimal organism may also be an exemplar of a Behe-type "genetic mousetrap." If the organism really is genetically minimal, the absence of any component will be fatal. And such a minimal organism will be noteworthy precisely because it will be a designed, laboratory artifact -- a drastic, artificial modification of a redundantly complex natural system. Thus it may be that biological mousetraps can result from deliberate design, but it will not be a case of art imitating nature!

Recent studies of *E. coli* bring to the fore another aspect of genomic redundant complexity which is relevant to the current debate. Observations have been made of proteins performing similar functions though with different genetic origins (Human Genome News, vol 8: 5-7). Such data provide evidence at the molecular level, of parallel, convergent evolution - supporting earlier observations of the same phenomenon (Smith *et al.*, 1983, p. 827) - and suggesting yet another way in which redundantly complex genetic and biochemical systems can manifest multiple causal routes to similar functional ends.

## **9. Conclusion.**

While there is much that we do not know about the biochemistry of living systems, it would appear to be premature to claim that there is a principled objection to the claim that the biochemical level of the biological hierarchy is itself a product of evolutionary processes. Behe claims that biochemical systems and processes manifest a species of complexity -- irreducible complexity -- that could not have evolved and must have been intelligently designed. We have shown, first, that systems satisfying Behe's characterization of irreducible biochemical complexity can arise naturally and spontaneously as the result of self-organizing chemical processes. Second, we have argued further that evolved biochemical and molecular systems exhibit *redundant complexity* -- this kind of complexity simultaneously accounts for the stability of evolved biochemical systems and processes in the face of even quite radical perturbations, for biochemical and metabolic plasticity, and, mainly as a result of gene duplication, for extant structures and processes to get co-opted in the course of evolutionary time, to serve novel functional ends.

In the end, Behe overestimates the significance of irreducible complexity because his simple, linear view of biochemical reactions results in his taking snapshots of selective features of biological systems, structures and processes, while ignoring the redundant complexity of the context in which those features are naturally embedded. Real biological systems are quite unlike economically designed engineering artifacts such as mousetraps. His case against evolution is a good example, in fact, of the perils of being "trapped" by a metaphor.

Of course, for some types of engineering problems, human engineers are not afraid to build in redundancy and back-up systems. Perhaps, Behe might want to argue, these sophisticated artifacts, with their redundant back-up systems, constitute a more sophisticated *design metaphor* by means of which to conceptualize nature. The trouble here is that naturalistic, evolutionary processes, notoriously, give rise to similar redundancies. And evolutionary processes do so without appeals to engineers of unknown identity and methods, be they cosmic, or merely alien, thereby commanding our attention on the basis of the scientific virtue of simplicity.

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