Notes on the Simulation of Evolution

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Abstract— **The simulation of evolution for the purposes of paramenter optimization has generally demonstrated itself to be a robust and rapid optimization technique. But there may exist more value in simulating evolution than simple parameter optimization. The optimizations of system behavior obtained through simulated evolution represent a potentially powerful autopoetic pathway to machine learning and self-organization. Indeed, the simulation of evolution may eventually prove to be the only practical path to the development of ontogenetic machine intelligence. As the complexity of the systems being evolved increases, the development of a proper philosophy of analysis and design becomes imperative. The designer of evolutionary algorithms must keep clearly in mind what is being evolved and what evolves only by consequence. Notes on the simulation of evolution are offered in four sections: 1) the basic nature of evolution, 2) the practical simulation of evolution, 3) common philosophical errors, and 4) that which remains to be accomplished.**

Simulated evolutionary optimization, as a machine intelligence technique, is still in its infancy, yet it is nonetheless a mechanism of machine learning that can reasonably be expected to continue to grow in importance and practical benefit. As the availability of massively parallel processors becomes increasingly common, the value of simulated evolutionary techniques will become increasingly apparent, if for no other reason than the natural match between the technique and the emerging technology.

The power of simulated evolutionary techniques cannot be easily exaggerated. The method has been repeatedly demonstrated to successfully find points of global optimality when other methods fail, often astonishingly quickly. While the process of simulating evolution is still very young, specific rules have become apparent and are easily stated.

I. The Basic Nature of Evolution

THE most salient rules of evolution, as they have come to be generally understood, are these:

- Evolution is not a force but a process [1]. The most overt attribute of the process is the accumulation of increasingly appropriate behaviors within an evolving lineage of trials.
- Learning is a matter of selectively retained behaviors, accumulated through stochastic trial-and-error, and is inherent to—and indistinguishable from—the evolutionary process itself [2]–[4]. Three distinct modes of learning are evident in natural evolution: 1) *phylogenetic learning* (learning in which adaptive behaviors are accrued within the lifetime of a phyletic lineage. The reservoir that accumulates phylogenetically learned behavior is the species' aggregate germline; the least unit of change in this reservoir is a base pair), 2) *sociogenetic learning* (learning in which adaptive behaviors are accumulated within the lifetime of the group. The reservoir of learned behavior is social culture; the least unit of change is a shared experience), and 3) *ontogenetic learning* (learning in which appropriate behaviors are learned through trial-and-error during the lifetime of an individual. The reservoir of learned behavior is aggregate neuronal and hormonal memory; the least unit of change is neurotransmitter titer and/or receptor site sensitivity).
- Darwinian evolution is an *optimization algorithm.* It is not a predictive theory, nor is it a tautology ([5] p. 519, [6] p. 112), as has often been claimed (e.g., [7], [8]). As in most optimization processes, the point(s) of solution wait to be discovered by trial-and-error search.
- Evolutionary optimization is a statistical process because it is a populational process.
- Reproduction in natural biota is generally very nearly replication, but it is not, nor can it be, perfect replication. In a thermodynamically "cold" universe, replication occurs without error. But in such circumstance evolution would be impossible. Error in replication is fundamental to the evolutionary process. However, at all temperatures greater than absolute zero, error in replication is inescapable and evolutionary optimization is inevitable in any self-replicating population constrained by a bounded arena.
- Darwinian evolutionary theory is composed of only five components: 1) a bounded arena, 2) a replicating population that must eventually expand beyond the bounds of the arena, 3) thermodynamically inescapable replicative error, 4) competition for space in that arena among the inevitable variants, and 5) consequential competitive exclusion of the lesser fit.
- Given self-reproduction, the processes and consequences of Darwinian evolution cannot be avoided in a finite, positively entropic universe.
- The Darwinian evolutionary algorithm exists without any sense of predetermined goal-directedness. The ultimate "goal of evolution" is determined solely by the shape and nature of the adaptive topography (the fitness function drawn as a "landscape") and is wholly external to the basic evolutionary process of variation and selection.
- Selection acts only to statistically cull the least appropriately behaving variants from the inevitable excess population. Selection never selects for any single trait in isolation of the behavioral whole. While the difference between the two processes may initially seem slight, the philosophies engendered by the differences in perspective are profoundly divergent in both their physics and their resultant effects.
- Behavioral error (as measured in current environmental context) is the sole quality sieved by competitive

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selection. Behavioral error is measured by the costs and consequences of incorrectly predicting forthcoming sequences of environmental symbol(s).

- Evolution optimizes behavior, not the encoding genetics *per se*, other than by consequence [11]. The specific nature and mechanisms of the genetical code are basically irrelevant to the process so long as they provide the necessary minimal complexity for a functional code set.
- Several naturally-evolved mechanisms operate to greatly increase the speed of optimization and general fitness of a population. They are: 1) the evolution of recombinant code (sex), 2) the evolution of individually adaptive behaviors (intelligence), 3) the evolution of hierarchical levels of modularity (cellular tissue differentiation, metamerism, and colonialism), and 4) the evolution of the processes of error repair and error expungement from the germline.
- The cell is the only engine of life. It is also the minimum atom of life; no smaller unit exists capable of self-sustained metabolism and reproduction.
- All organisms large enough to be seen with the unaided eye are composed of complexly interacting colonies of (nearly-) genetically identical cells. The concept of the individual is somewhat deceptive. All higher levels of cellular organization acquire the attributes of a colony where differentiated units are recruited into synchronized behaviors, operate to a common purpose, and are evaluated as a whole.
- Only three forms of knowledge reservoirs have been evolved within natural circumstance. They are: 1) the reservoir of phylogenetic knowledge implicit in germline DNA, 2) socially communicated and remembered experience (culture), and 3) the hormonal and neuronal memory of individuals.
- The differences between accrued knowledge (information) and behavior are explicit. Stored (genetic) information encodes individual trial (phenotypic) behavior. Behavior is evaluated as to its fitness only in current environmental context. Unexpressed code has no intrinsic value in and of itself.
- Because of the extensive modularity characteristic of complex organisms, behavioral errors can be often attributed to specific subsystems ("traits"). Evolution optimizes the total system error of the species by minimizing the costs of specific behavioral errors in the general order of the costs those subsystem errors incur. Selection operates differentially on specific portions of the behavioral response surface proportionally to the magnitude of the "trait" error and the specific cost of that error (mosaic evolution).
- The twin phenomena of *pleiotropy* (one "gene" affects many traits simultaneously) and *polygeny* (many discrete coding structures combine to generate one "trait") operate to create virtually continuous behavioral response surfaces in highly polyparametric state spaces, no matter how discrete the individual coding components may be.
- Pleiotropy and polygeny exist in all complex information systems and cannot be completely eliminated from any such system. The evolution of non-interacting code, either by random selection or purposeful design, is virtually impossible. This inevitable result renders any philosophical attempt to assign specific fitness values to a single component or coding structure fallacious.
- Explanations of the role and purpose of any single component—or combination of components—in isolation of the whole, from the "bottom-up," are generally in error. Such explanations often demand philosophical recourse to "emergent properties." Such philosophies generally exhibit a pronounced tendency to reverse cause and effect and often significantly misrepresent the evolutionary process.
- The simplest, most probable (thermodynamically "cheapest") method to quickly build complex behavior is to reuse and reoptimize existing code for secondary purposes. This phenomenon, which is commonly called "ditto programming" or "code stitching" in engineering, is called "preadaptation," "exaptation" [12], or "metamerism" [13]–[14] in evolutionary biology.
- Complexity does not evolve for complexity's sake, but for the purposes of continuously increasing a refinement in the appropriateness of behavioral responses. As competition becomes increasingly intense between contending lineages, as it must in all stable environments, selection increasingly distinguishes among subtle differences in behavioral appropriateness. Simple coding structures cannot generate intricate or subtle behaviors.
- As complexity builds, some fraction of the code becomes immutable, merely due to its critical location in an evolved coding hierarchy. Ancient code becomes a foundation on which all further evolution is built. Any modification of this early code generally has pronounced effects that ripple throughout the expression of the whole of the evolved coding structure. Because of this hierarchical layering of code, early coding structures often become critically placed "symbols" in an internally tiered system-behavior alphabet.

II. Simulating Evolution

Although the informational physics that governs the evolutionary process may ultimately prove to be quite simple, the structures built by evolutionary trial-and-error are enormously complex. Because of this complexity, evolutionary theory has been plagued throughout its history by misrepresentation and misunderstanding.

The philosophical problem that has historically bedeviled the development of evolutionary theory has lain in the choice of a proper approach to the analysis of complexity. Much of the confusion surrounding the interpretation of the evolutionary process has been the result of a single subtle, and seemingly innocuous, choice in philosphical perspective. The choice lies between: 1) celebrating the nature of specific components (genes, neurons, etc.) or processes (recombination, genetic representations of an individual, etc.) and assigning specific values to these attributes in isolation of the whole, or 2)

emphasizing those processes that optimize and evaluate the whole of the evolving structure.

The proper choice of perspective remains essentially unresolved today. It is possible to find authors that forcibly argue either view. The choice of perspective has not proven to be an easy one, or one that has been always consistently maintained, even within the thoughts of a single author, but it will be argued here that: 1) it is possible to make the choice transparently clear, and 2) that the correct choice is essential to the efficient exploitation of evolution for engineering purposes.

The philosophical celebration of individual components or processes in isolation of the whole almost always reverses the true chain of causation and profoundly misrepresents the process of evolutionary optimization, especially when the attendant philosophies are extrapolated. The nature of this very fundamental philosophical error is discussed in detail in Section III. This misplacement of perspective has persistently recurred during the development of evolutionary theory and has been the source of some of evolutionary biology's most contentious arguments. The same arguments are virtually certain to similarly become part of the engineering discussion.

But if the physics of evolution is well understood and the chain of causation is properly represented, the process of evolution can be stated in rather simple terms and can be simulated for engineering purposes. Given the complexity of evolved structures, it is reasonable to be somewhat surprised that evolution appears reducible to so very few rules.

Darwinian evolution is a strictly physical process. One of the most accurate derivations of Darwinian evolutionary theory was one of the earliest: Boltzmannian thermodynamics. Boltzmann was deeply moved by the idea of simple selection rules operating against populations that possess intrinsic variation. He wrote that if the 19th century were to be given a name, it would be called "the century of Darwin" [15]. Shannon [16]–[17] later reinterpreted Boltzmann's entropy, defining "information" as unexpected variation. Shannon's information measure was defined more as a metric of surprise than disorder. In that, a philosophical circle was completed. Evolutionary optimization is, on its surface, a rather simple thermodynamic process where the agent of selection (competition in a bounded arena) operates to minimize the behavioral inappropriateness (surprise) of the trial variants.

This inherent simplicity allows for a reasonably accurate, first-order simulation of evolution for engineering purposes. Simulated evolutionary optimization algorithms are normally implemented in the following manner:

Step 1: An initial population of "trials" is chosen at random. The size of the population, N, may range over a broadly distributed set but is generally much larger than 1.

Step 2: The population is replicated, with error, such that each parent "trial" generates c progeny, where c is a small number, normally ranging between 1–1000. The form of the replicative error imposed on the behavioral response must be stochastic and must range in intensity from very minor to very substantial, as continuously as possible to be maximally effective. If the mutational step size is bounded and/or fixed, entrapment is generally inevitable on complex topographies.

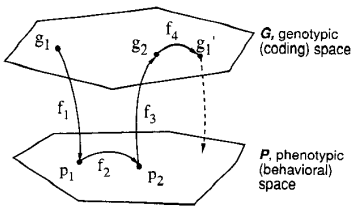

Fig. 1. The evolution of a population within a single generation (after [17]).

Step 3: The quality of behavioral error is assessed for all members of the population, parent and progeny. One of two conditions is generally implemented: 2) the best N are retained to reproduce in the next generation, or 2) N of the best are probabilistically retained. In either manner, the population remains size-constrained, and ultimately the competitive exclusion of the least appropriate ("least fit") is assured.

Step 4: The process repeats by returning to Step 2. A halt to the sequence is declared when either a predetermined quality of solution has been achieved or a set number of iterations have been exhausted.

Any number of minor variations of this basic procedure are possible, and many have been implemented (e.g., [9], [10], [23], [24], [36], [37], [39], [47], [54]), but these few steps are generally characteristic of all simulated evolutionary procedures.

A. The Formalization of the Process

Two state spaces are inherent to the evolutionary process: a genotypic (coding) state space, **G**, and a phenotypic (behavioral) space, **P** (Fig. 1). Similarly, two alphabets are also defined; an input alphabet set of environmental symbols, **I**, and an output set of behavioral responses, **Z**.

The evolution of a population within a single generation is schematicized in Fig. 1 [17]. Consider a population of genotypes, $g_i \in G$. There exists a mapping function f_1 such that each g_i is translated into a phenotype p_i. The mapping is inherently many-to-one because there exists an infinity of genotypes which may result in an identical phenotype; an infinite set of unexpressed code may exist in g_i.

However the development of a complex phenotype is rarely, if ever, determined solely by its genetics. In naturally evolved, multicellular species, the translation of g_i is generally extensively mediated by cell-local environmental context. With very few exceptions, identical code exists in every cell of a multicellular organism. However, only a fraction of the code is normally expressed in any one cell phenotype. Instead, the development of the specific cell phenotype results from the expression of ca. 50–75% of the total code present in the cell [31]. Cell development that is sensitive to local conditions is termed *epigenesis*. The evolution of context-dependent development offers the lineage the highly advantageous capacity of manufacturing of a variety of cell and organismal phenotypes, each appropriate to specific local environments, by creating a multitude of alternately expressable routines.

The first mapping of Fig. 1, *epigenesis,* incorporates these rules of genetic expression, growth and development, under local context and may be written as:

$$f_1 : \mathbf{I} \times \mathbf{G} \rightarrow \mathbf{P} \tag{1}$$

Epigenesis is a dominant theme at the level of the cell. All tissue differentiation during embryogenesis is the result of locally determined developmental pathways. But epigenesis is not unique to the cell; higher-order organismal-level developmental epigenesis also exists and is called *phenotypic plasticity.*

This developmental plasticity is relatively common in plants, which, by their nature, must develop and grow in place. The extent of phenotypic plasticity in plants is such that individuals of the same species that have grown in different environments have occasionally been misclassified into different genera. Phenotypic plasticity is more subtly expressed in animals, but it is not uncommon. Simple examples are the determination of wing color in some moths and butterflies and gender in turtles, each of which is influenced by the temperature at which development occurs. But one of the most pronounced examples of such developmental plasticity occurs in the control that many species of ants and bees exert over the manufacture of their various highly polymorphic castes. The signalling substances contained in royal jelly alter the developmental sequence of worker bee larvae and transform them into the highly differentiated queens.

The highest level of context-sensitive phenotypic development, which occurs only in animals, is wholly behavioral and is termed either *habituation, conditioning,* or *imprinting.* Although classified as learned responses, and thus attributes of ontogenetic intelligence, each represents a form of context-sensitive development. Imprinting is the most exaggerated expression of this form of learning in that it is wholly ineradicable and thus no different than phenotypic plasticity in its effects once it is fixed-in ([49] pp. 262–314).

Much of the reason why evolution may prove to be so readily describable by so few rules is that only a very few informational patterns, once engaged, appear to be repeated throughout a hierarchy of discrete levels of organization. Epigenesis is one of these patterns. Iterated patterns of differentially expressed behaviors permeate the design of all complex biota.

The second mapping, *selection,* describes the processes of selection, immigration, and emigration of individuals within the local population:

$$f_2 : \mathbf{P} \rightarrow \mathbf{P} \tag{2}$$

The effects of selection occur only in $\mathbf{P}$ and have no direct effect on the coding structures in $\mathbf{G}$, other than by the consequences of selective survival.

The third mapping, *representation,* describes genotypic representation within the population prior to reproduction:

$$f_3 : \mathbf{P} \rightarrow \mathbf{G} \tag{3}$$

The fourth mapping, *mutation,* incorporates the rules of random and directed coding alterations (including repair and recombination):

$$f_4 : \mathbf{G} \rightarrow \mathbf{G} \tag{4}$$

Only through the repeated selection of appropriate behaviors in $\mathbf{P}$, generation after generation, does a learned reservoir of appropriately functional code accumulate in $\mathbf{G}$. The evolution of a phyletic lineage proceeds through the indefinite repetition of these four mappings.

Lewontin ([17] p. 15) has warned against too facile a view of evolution as composed of two separate, parallel systems of processes, one genotypic, one phenotypic. That separation is "illusory." The two state spaces are tightly bound by the mappings described above. But, it is also important to clearly delineate which part of the evolutionary process appears in which state space. Confusing the attributes of the two state spaces lies at the root of much of the confusion that permeates evolutionary theory.

B. The Language-Like Nature of Evolutionary Optimization

Evolutionary optimization is not characterized by simple combinatorial optimizations. Rather, it is intrinsically composed of problems that minimize the costs of mispredicting sequences of environmental stimuli. Misprediction of a forthcoming event (surprise) is generally costly, if not occasionally lethal. The syntactical relationship that must inevitably evolve between the increasingly acute perception of strings of external stimuli and the evolution of appropriate responses assumes the nature of a language, even when implemented only as a simple reactor to such stimuli.

Two attributes characterize all complexly behaving systems: a combinatorial, stimulus pattern recognition component and a time-sequential, memory component. Both attributes are present in a simple mathematical model, finite state automata (FSA's). Finite state automata, which were devised to mimic the nature and behavior of the digital computer [18]–[19], represent an especially simple but appropriate description of the behavior of natural biota. FSA's are described by a five-tuple, $\langle \mathbf{Q}, \mathbf{I}, \mathbf{Z}, \delta, \omega \rangle$, where $\mathbf{Q}$ is the set of all internal states, $\mathbf{I}$ the input alphabet of environmental symbols, $\mathbf{Z}$ the output symbol set, δ, a next-state mapping function, such that:

$$\delta : \mathbf{I} \times \mathbf{Q} \rightarrow \mathbf{Q} \tag{5}$$

and ω, an output mapping function, such that:

$$\omega : \mathbf{I} \times \mathbf{Q} \rightarrow \mathbf{Z} \tag{6}$$

The five-tuple partitions itself naturally into the two state spaces of Fig. 1, $\mathbf{G}$ and $\mathbf{P}$. The three-tuple, $\langle Q, \delta, \omega \rangle$, the *genome,* is a component solely of the informational state space, $\mathbf{G}$. The remaining two-tuple, $\langle \mathbf{I}, \mathbf{Z} \rangle$, *stimulus-response,* is an attribute only of the behavioral state space, $\mathbf{P}$.

The behavioral error of a single phenotypic FSA may be assessed as the accumulating sum of the magnitude of the phenotype's misprediction of current environmental symbols:

$$E = \sum |i_{t+1} - z_t| \tag{7}$$

Behavioral competence rapidly evolves, not merely as a matter of predicting the next symbol as a simple conditional

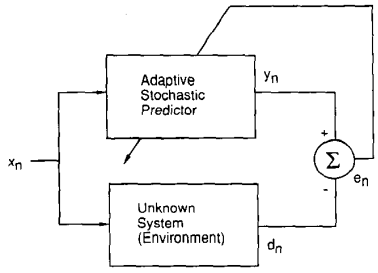

Fig. 2. An adaptive stochastic predictor/filter (after [46]).

probability, $\Pr(i_{t+1}|i_t)$, dependent only upon the previous symbol, but on the previous m+1 symbols as a Markov process:

$$\Pr(i_{t+1}|i_t, i_{t-1}, i_{t-2}, i_{t-3}, \ldots, i_{t-m})$$

If no correlation exists between environmental symbols, higher-order evolution becomes impossible. But it is the nature of the physical universe that certain symbols strongly portend subsequent symbols, and strings of specific signals are often quite specific in their portent. The segregation of such strings from random noise and the subsequent increase in the length of these predictive strings is the essence of evolutionary adaptation.

C. Topology Evolves First

Figure 2 is representative of all self-adapting systems. Although drawn in standard engineering form as a stochastic filter/predictor, Fig. 2 is nonetheless also an eloquent description of the Darwinian optimization process. So long as the adaptive process is stochastic, it is intrinsically populational. Survival of the contending variants within the stochastic population is mediated solely through competitive selection in a manner such that the overall populational error, e_n, is minimized. The control that natural selection excercises over the effects of random variation within the population is a negative feedback process, composed of a constant comparison between the present representation of the world and new information coming in from it, and a constant adjustment in the population's (system's) behavior in light of that comparison. The end result, adaptation, tends to emulate the result of deliberate, conscious design ([21] pp. 169–174, [55] p. 17, [56] pp. 21–27, [57] p. 4).

The population of trial variants implicit in Fig. 2 are not necessarily simple machines. Indeed, the common situation is that they are populations of rather elaborate and complex circuits. It is inherent in the nature of any complex system that not all of its components are simultaneously active. Behavior flows through various subsets of the system's networks in response to specific external stimuli. The optimization of overall system behavior, whether the implementation was accomplished mechanically, electronically, biochemically, or mathematically, is a matter of the proper choice of two separate aspects of design: 1) the topology of flow, and 2) the selection of the values of the system's constituent rate-determining components.

The majority of evolutionary simulations conducted to date have unfortunately not been fully representative of the nature of complex systems, either naturally evolved or purposefully designed. Most applications of simulated evolutionary techniques have been applied to optimization problems where either 2) the topology was predefined and invariant, as in the case of neural net weight determinations (e.g., [23]) or in multi-pole, multi-zero filter coefficient optimizations (e.g., [46]), or 2), in distinct contrast, where rate-determining coefficients were made fixed at the time of random generation and the topologies of the trial variants were randomly varied (e.g., [37]). Because each of these conditions fix one of the two central attributes of design common to all stimulus-response machines, both tend to understate the true nature of evolution in complex systems.

When both the topological organization and component values of the phenotypic trials are freely allowed to vary, virtually all initial gains are made in topological reorganization. The reason is straightforward. A simple network can produce behaviors of only limited complexity. In order to properly evolve appropriately subtle responses, a specific minimum system complexity is necessary. In most circumstances, component values need to be only coarsely correct to evaluate the merits of various alternative topologies.

However, after a series of early, initial gains in fitness have been realized, topological reorganizations wane in their capacity to provide further significant advances in system designs. Indeed, most topological reorganizations will either come to be neutral (the section of reorganized topology is unexpressed in the current environment) or increasingly deleterious. Modification of the logical topology generally results in a pronounced alteration of system behavior and becomes an increasingly severe mutation as optimality is approached. End-game transformations are almost solely composed of component value changes.

D. Tunneling

Complex adaptive topographies are often pocked with local optima. The primary escape mechanism from local optima is populational polymorphism, resulting in a form of statistical "tunneling," which is not dissimilar to quantum mechanical tunneling.

The physics implicit in any mathematical gradient-search technique that advances by assessing the local contours of the adaptive topography is structurally different from that characteristic of evolution. Mutagenesis occurs without respect to the shape of the adaptive topography and is restricted wholly to the coding state space, $\mathbf{G}$. Only after $g_i \in \mathbf{G}$ has been mapped into $\mathbf{P}$ are the $p_i \in \mathbf{P}$ phenotypic trials mapped onto the optimization topography and evaluated.

Sufficient experience has now been garnered to suggest that all strongly-determined, contour-dependent numerical optimization methods tend to work acceptably well on some adaptive surfaces, but exhibit a pronounced tendency to stall in indefinite oscillation or become entrapped in local optima on others.

These stalls are caused by a highly-determined correlation between parent and child trial vectors in which some aspect of the adaptive topography (e.g., the gradient of the topography) plays a role in the specification of the trial progeny (generally, direction and step size). But such deterministic linkages between the trial progeny and the slope and contours of the adaptive topography are not characteristic of the manner by which evolution proceeds. The mutagenesis guaranteed by replicative error in **G** generates a continuum of very fine- to very large-grained variation in **P**. That phenotypic variational continuum is functionally necessary for the escape from local optima. The correlation between parent and child trial vectors under evolutionary optimization is often strong, but it cannot be absolute. But most especially significant, the slope, current position, or contour of the adaptive topography plays no role in the mutagenesis of genotypes in natural situations. This simple observation is the reason why evolutionary search algorithms are intrinsically resistant to entrapment on contoured surfaces. The optimizing process of trial (phenotype) generation is wholly decoupled from the contours of the adaptive topography.

Large-scale random variation creates an escape mechanism by means of populational "tunneling," as diagrammed in Fig. 3. The novel existence of a single phenotype in the region to the right of the dotted line will draw the remainder of the population to the right-lying well. The new phenotype will exhibit an error score less than all other members of the population and is thus offered a greater statistical chance of differential reproduction. The survival of a single novel variant strongly implies the survival of other similar variants and, most especially, their progeny. Because the adaptive topography is greatly steeper to the right of the line, the remainder of the population is very rapidly "drawn through" an imaginary tunnel. No more than a limited number of phenotypes may survive at any epoch, as dictated by the size of the competitive arena, and for a brief period of time, both morphotypes will exist in the population. Very soon however, the population will again become monomorphic. All surviving phenotypes (and genotypes) will come to occupy the region of the state space to the right of the line.

Perhaps surprisingly, the height of the intervening barrier is immaterial to the tunneling process. It is not the height of the barrier that determines the rate of escape from a local optimum but its width relative to current populational variance. Under Gaussian-driven mutation, no phenotype is impossible; thus, global solutions in a finite state space topography are guaranteed in infinite time. However, if the range of phenotypic variation should be constrained to a few fixed step sizes, local optima will exist from which no escape is possible. One or more intermediate phenotypic forms must lie at a higher error point on the extended intervening barrier.

E. Probabilistic Selection

The capacity to escape from local optima is fundamental to any search algorithm if the global optimum is to be discovered. One of the more readily advocated mechanisms to accomplish this escape is probabilistic selection. Such

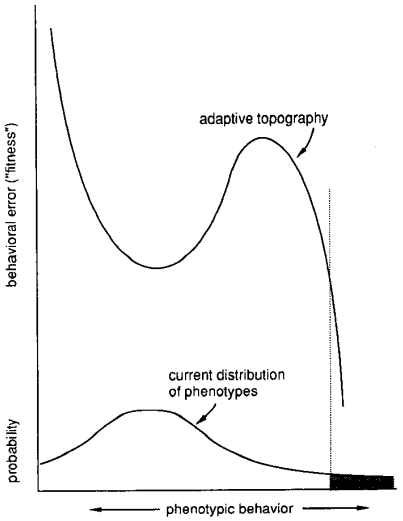

Fig. 3. The tunneling escape of a population from a local optimum.

selection exists when a population of size **N** is retained with reduced consideration given to fitness rather than stringently selecting only the best **N** phenotypes to be progenitors of the next generation. The advantage ascribed to probabilisitic selection is that phenotypes that would not exist under intense selection may otherwise survive, thus allowing a population to "walk" out of a local minimum.

Probabilistic selection also decouples the distribution of phenotypes from the contours of the adaptive topography; in that, it is similar to tunneling. Although some selection noise exists in all natural situations, the advocacy of probabilistic selection should however be conducted with caution. This form of decoupling is obtained only through a reduction in the intensity of selection and is not equivalent to tunneling. Complete decoupling from the topography occurs only when selection pressures have been fully released. At this extreme point, evolution is no longer progressive. Rather, it has become wholly entropic. Any phenotype becomes as acceptable as any other and survival is simply random.

The advocacy and use of probabilistic selection is not a black-and-white choice. Natural selection pressures are occasionally abated during catastrophes. These episodic releases of selection pressures often create a "diversity pump" [41], [47] by allowing a variety of types to survive for a period of time unchallenged by the sieve of intense selection. And clearly shallow contours on the topography can be more easily traversed when the intensity of the selective filter is reduced. But deeper wells require a significantly greater relaxation in selection pressures.

This is not generally a natural condition. With the noted exception of catastrophes, selection pressure tends to remain intense in natural populations. Selection acts not only as a constant editor active in the acquisition of new characters and behaviors, but also in the maintenance of existing structures. Once selection pressure has been relaxed in a complex system, evolved subsystems rapidly atrophy in proportion to the release of selection pressure. A natural example lies in the eyes of cave

animals. Individuals with imperfect eyes are not eliminated from the population and eyes often rapidly disappear or become dysfunctional in such species ([5] p. 502). Weismann wrote in 1893, "In my opinion every organ is kept at the peak of its conformation only by continuous selection" ([51] p. 51).

F. The Acceleration of Optimization

While the necessity of an infinite mutator is clear, broadly scattering progeny greatly slows the evolutionary search and wastes phenotypic trials. Decreasing the variance of a Gaussian mutator function, especially as optimality is approached, dramatically accelerates the search.

Gross evolutionary optimization generally occurs quite quickly, the speed of which is proportional to the steepness of the adaptive surface's wells. But as optimality is approached, the surface of the adaptive topography flattens and phenotypic (trial vector) wastage becomes an increasingly pressing issue.

Although the process may initially appear counter-intuitive, the stabilization of genetic information accelerates evolutionary optimization by reducing the effects of unconstrained variation. Collapsing the variance of a normally-distributed mutagenesis function proportionally to the error (fitness) score increases the density of trial vectors by a factor of $(\sigma_{t'}/\sigma_t)^n$, where σ_t is the characteristic variance of the state vector at time t and n the rank of the trial vector. Because n is often very large (50 000–100 000 in humans), small changes in σ produce large volumetric changes in the state space which is to be probabilistically evaluated [20].

Two forms of population-culling selection were identified by Darwin, *natural* and *sexual* selection [21]–[22]. Natural selection is the sum of all competitive culling phenomena that are *extrinsically* imposed on the evolving phyletic line. But the second form of selection, sexual, is evolved *within* the lineage.

Sexual selection, which Darwin saw as a "milder" form of natural selection ([21], [22]) reduces populational variance by excluding defective breeders from the germline (Fig. 4). Primary congenital defects often have significantly debilitating effects on their carriers. The population-genetic effect of intense and prolonged male-male contest is a (significant) reduction in populational variance. By reducing the intrinsic variance of a reproducing population, the effect is one of greatly 1) accelerated optimization, 2) minimized phenotypic wastage, and 3) improved overall populational competitiveness.

Variance is also similarly reduced through the evolution of cell-internal genetic repair mechanisms. But repair is constrained, by its very nature, to be a less precise process. The evolution of any repair algorithm, naturally evolved or purposefully designed, demands that errors come to be recognized in an artificial manner: by measuring the quality of the method by which the code has been packetized, not in the actual worth of the message contained within the packet. This attribute is characteristic of all error-correcting mechanisms, whether they are the information-assurance procedures common in computers, such as cyclic redundancy codes, hamming codes, and parity checks or are naturally evolved processes such as long- and short-patch excision common to the repair of

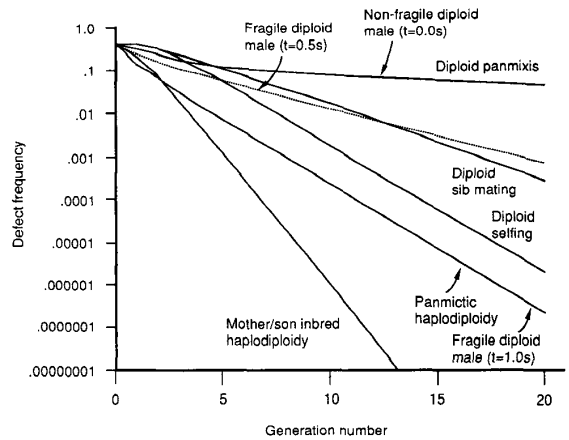

Fig. 4. Theoretical rates of defect expurgation from natural populations as a function of the form of mating system employed (after [35]).

DNA. Repair mechanisms are intrinsically isolated from any assessment of the content, and most especially the value, of the messages contained within the coding packets. Nor can a finite set of error repair algorithms detect and repair all forms of error. Some error will pass uncorrected simply because it will lie outside the province of the error-correcting algorithm.

Strong evolutionary pressure must exist to evolve both mechanisms of variance reduction. The efficiency of the defect expurgation process does not need to be pronounced to be effective. The effect of detecting and excluding error from the coding germline is multiplicatively regenerative, generation after generation, honing species-specific variances to levels at or near environmental noise.

The practical advantages in accelerating the rate of optimization by simulating this reduction in variance as optimality is approached have been repetitively demonstrated in simulated evolutionary algorithms (e.g., [9], [24], [54]).

G. The Acceleration of the Simulated Annealing Algorithm

The evident similarities between the simulated annealing algorithm [25] and the various evolutionary optimization algorithms are not coincidental. Not only are the theories derived from a single source philosophy, the natural processes are fundamentally equivalent. Boltzmannian thermodynamics is selection imposed on a population of variants.

Simulated annealing is a stochastic search algorithm modeled around the cooling coalescence of a crystal. The algorithm has proven to be successful because it imposes a constrained random search on a fitness topography. It also inherently incorporates the very powerful accelerating mechanism of a constantly reducing variance. The algorithm operates by defining a beginning temperature, T_0, and an extrinsic annealing schedule specifying the rate of temperature reduction. In effect, the annealing schedule specifies the rate of variance reduction. As $T \rightarrow 0$, stochasticity is removed from the system. At $T = 0$, the process is said to be "quenched."

The criticisms most commonly directed against the simulated annealing algorithm are: 1) if too low an initial temperature is chosen, the process may settle into a local optimum

and quench itself there, 2) if too high a temperature is chosen, the algorithm is slow, and 3) although resistant to entrapment, the algorithm can become occasionally entrapped ("frustrated" [25]).

The principal problems with the method as it was originally formulated are: 1) that only a single point is retained at the end of each iteration. This single line of search increases the probability of entrapment in an isolated local optimum, and 2) that reductions in variance are tied to an extrinsic cooling schedule unrelated to the present quality of solution. These problems can be easily rectified by 1) significantly increasing the trial population size, and 2) coupling reductions in variance to quality of the present solution by reducing system variance, σ^2, proportionately to the current epiphenotypic (populational average) error, $E(t)$, at time, t.

Increasing the size of the effective population, N_e, maintained at each generation works to increase both the rate of search and significantly increases resistance to easy entrapment. A population $N_e >> 1$ allows for potentially N_e independent, simultaneous (polyphyletic) searches of the state space.

Polyphyletic searches greatly increase the opportunity to locate and descend the deepest minimum error wells. Evolutionary experimentation is inherently a parallel process. The larger the effective population, N_e, at each generation, the more comprehensive the exploration (the greater the "sense") of the state space surrounding the current epiphenotype at each generation. This greater exploration of the adaptive topography feeds back into the population of trial vectors, and thus shallow local optima that might otherwise entrap a lineage of single trials for a time are traversed unnoticed.

While these modifications in procedure make many of the remaining differences between the simpler forms of simulated evolutionary algorithms and simulated annealing trivial, there also exists a profound difference between the two approaches that cannot be overstated. Optimizations obtained through simulated annealings are accomplished only in the phenotypic state space, **P**. No underlying coding state space, **G**, exists in a simulated annealing. The consequential difference of this very fundamental disparity in structure is that no reservoir of learned behavior can come to exist in any procedure similar to simulated annealing. While both approaches can rapidly optimize complex functional behaviors through stochastic searches, it is the nature of evolution to build large and extensive libraries of learned knowledge in **G**.

The simple existence of these libraries of evolved code represents not only an enormous difference in the philosophical approaches of the two algorithms but also in the range of applicable problems. Any approach to stochastic optimization that follows the philosophy implicit in simulated annealings must always be relegated to rather simple function optimizations; the process is Markovian and memoryless. In contrast, simulated evolutionary algorithms, simply because of the presence of distinct coding structures that evolve consequentially, hold within their structure the promise of the eventual evolution of self-organizing, ontogenetic machine intelligence.

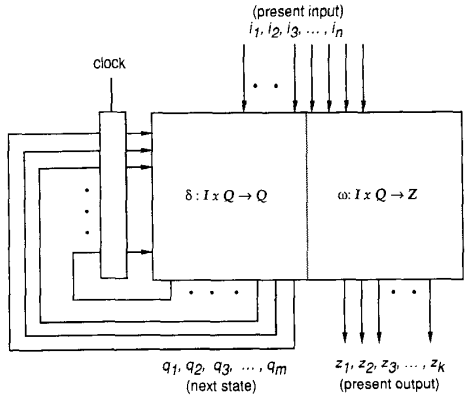

Fig. 5. The minimum hardware configuration required to realize a stimulus-response machine of any finite complexity, either of fixed or evolvable behavior (after [47]).

H. Minimum Evolutionary Hardware

If the mappings presented in (1)–(6) are an accurate description of the evolutionary process, then there exists a minimum hardware set that will implement those mappings. That minimum set is composed only of a mass of memory, a bank of m flip-flops, and a clock (Fig. 5). The behavior of any finite-state machine may be created (or evolved) in this architecture, regardless of complexity. If the machine to be modeled is a pattern recognition device (e.g., a neural net), then only the right-hand part of the memory is necessary and the stimulus-response mapping merely becomes:

$$\omega : \mathbf{I} \rightarrow \mathbf{Z}$$

But if time-sequential behavior is to be incorporated, as it must in any general machine, the entire structure is necessary.

The memory plane may be built as standard digital computer memory, consisting of $m + n$ address lines (current stimulus and state) and $m + k$ output lines (next state and present reponse). Evolution would proceed with the creation of cN_e such machines at every generation, where c is the average number of mutated progeny, placed in competition with their parents such that only N_e survive.

III. Common Philosophical Errors

Darwinian evolution, at its simplest, requires only variations in the coding structures in state space G and the selective culling of subsequent trial expressions within P. The very fundamental question that arises is how to properly interpret not only the result, but also the process.

The interpretation of the evolutionary process has not proven itself to be inherently simple. Systems of enormous complexity have been evolved. This complexity presents any number of facets, each of which may be anyalyzed in its own right, in piecemeal fashion. But explanatory hypotheses that tend to emphasize only a single facet of evolution are prone to misrepresent the process, and most especially misconstrue causation.

Most troubling has been the elucidation of purpose. In distinct contrast to engineering, where purpose within a design

is taken for granted—and where the author of a design may perhaps still be available for questioning as to his reasons and movtivations for specific details, no such recourse is possible in naturally evolved systems. Indeed, the degree to which to even recognize the nature and extent of purpose within naturally evolved biota has proven to be one of biology's longest and most fundamental internal debates. Haldane once quipped, "Teleology [the study of purpose] is like a mistress to a biologist; he cannot live without her but he's unwilling to be seen with her in public" ([50] p. 392).

But purpose clearly exists in the designs produced by evolution and the reintroduction of purpose into the biological discussion has been championed by biologists such as Pittendridge, Lorenz, and Mayr. Pittendridge [52] renamed and redefined the study of purpose in evolved structures to be *teleonomy* in order to draw as sharp a distinction between it and the mysticism of an older teleology as currently now exists between astronomy and astrology ([49] p. 29).

The purpose of an eye or a wing is self-evident. The problems lie in the analyses of far less obvious structures where derivative interpretations are often extensively colored by the type of biology being conducted. A physiologist will often speak in sentences that inherently presume a profound purposefulness in the structure(s) under study, while most molecular geneticists would be extremely reticent to resort to such ascriptions.

The reasons for the inherent differences in perspective specifically lie in the two state spaces, **G** and **P**. Adaptation through iterated selection occurs only in **P**. Physiological structures are the tangible system behaviors evolved within the state space **P**. The genetical codings that come to exist in **G** evolve only by consequence. All sense of direct causality between the code and its effects is lost in the web of interactive effects that connects a highly pleiotropic genotype to a very polygenic phenotype. Causality becomes extremely diffuse, and thus especially intractable to simple analyses in this second state space.

The same difficulties prove similarly true in artificially evolved systems. A population of machines, composed of the architecture of Fig. 5, appears capable of evolving the behavior of any finite-state machine, regardless of complexity. But it requires very little experience to discover that explicit if-then-do rules of learned behavior will not come to exist in the randomly evolved coding structures of Fig. 5. Because certain coding patterns will be used for a variety of purposes (that is, logical flow will pass through them many times), the patterns will defy simple analysis. Rather, even as evolved behavior approaches perfection, the bit patterns in the memory plane will become a hodge-podge, with few, if any, simple causalities easily deducible.

The search for simple, heuristic rules has been the subject of considerable and frustratingly fruitless effort in artificial intelligence work. Equally frustrating have been attempts within an earlier biology to tie specific "genes" to specific "traits." It is human nature to reify abstract processes, but such reification is often self-defeating because it need be only slightly wrong to profoundly misrepresent the process at hand. The rule that must be kept clearly in mind is: Whenever code and behavior are separate entities, as they almost always are, functional behavior is the quality directly optimized by selection, not the underlying code.

A second confounding feature of evolution is that adaptation in **P** evolves as a unit whole. Traits and behaviors in a complex behavioral structure do not exist as independent and isolatable rules. "The organism is not an aggregrate of 'unit' traits or characters or qualities. Traits, characters, and qualities are not biological units; they are abstractions, words, semantic devices that a student needs in order to describe and communicate the results of his observations. A trait has no adaptive significance in isolation from the whole–Talking about traits as though they were independent entities is responsible for much confusion in biological, and particularly in evolutionary, thought" ([27] p. 64).

Functional interactivity permeates all complexly evolved coding structures, mechanical or biochemical. The change of a single bit may have unanticipated effects that extend throughout the behavioral repertoire of the complete machine. Or, the change of a single bit may have no effect at all. Evolutionary optimization is a pragmatic and opportunistic process, a matter of adopting whatever functional variant works.

The fundamental philosophical choice that must be made at the outset is whether to emphasize the mechanisms of the coding structure or, in contrast, the processes that maximize total system behavior optimization. If only one gene encoded one trait, and if the traits were independent, the choice of perspective would be irrelevant. Optimizing one state space would automatically optimize the other. But if the interactions between "traits" and "genes" are pervasive and complex, then only one of the two spatial surfaces may be optimized. If "genes" (subroutines) are perfected as isolated rules, acting in their own best interests to maximize their own individual survival, then the behavior of the phenotype (system) must inevitably become a non-optimal collection of individual behaviors. But if the phenotype is "perfected" as a unit whole, the underlying genetics must, by necessity, become a melange of interwoven, overlain, and duplicated code. Purpose does come to exist in evolved structures, and that purpose can be deduced. Mayr ([50] p. 399) has written, "It is no exaggeration to claim that most of the greatest advances in biology were made possible by asking 'Why?' questions." But purpose only clearly exists in the adaptive structures and behaviors evolved within state space **P**. "Why?" is a question that can only be asked in a meaningful manner in **P**.

Neither Darwin nor Wallace fundamentally understood genetics other than as a general description comprised of the "laws of inheritance." But a detailed understanding of genetics was not necessary to correctly deduce the nature of evolution. Indeed, it could profitably be argued that such an understanding would have only complicated the discovery of the process. Darwin described in detail the process of evolution and the resultant system behaviors embedded in **P**: ethologies, morphologies and physiologies—and their purposes. When the philosophical perspective is constrained to the clear chain of causation resident in **P**, "...the designs developed by

evolution are so similar in principal to those that would be reached by a conscious designer, ...it seems reasonable to suggest as a general approach to biological problems that the investigator should ask himself what are the essential functions involved and how might a designer provide for them" ([57] p. 4).

A correct philosophy of design is fundamental to all future engineering progress if the exploitation of simulated evolution is to succeed. Indeed, a correct understanding of the physics in force, of what's actually occurring, is arguably more fundamentally important to engineering than it is perhaps to biology itself. Analytical observations are, to a great degree, self-correcting. But the synthetic design process characteristic of engineering will collapse under the weight of its own misunderstanding. If the adopted philosophy is incorrect, design failures are assured.

Five simple, but fundamental errors that have plagued the proper analysis of function and purpose in evolutionary biology are discussed in the following few paragraphs. A few of these errors are already beginning to be repeated in the engineering literature.

A. Error 1: Confusing a Defect with a "Trait" in Complex Systems

Among the most common errors in evolutionary biology has been the confusion of a point defect with a selected trait. A portion of this error arises from a misinterpretation of the nature of redundantly encoded subroutines.

Mendelian "genes" are virtually always defects. Mendel's classic "yellow" and "wrinkled" peas are the consequences of single point recessive defects in a twice-redundant (diploid) coding structure. Under diploidy, with very few exceptions, two copies of all genic subroutines are maintained in every cell of the organism.

The information resident on all paired chromosomes is actively translated when both copies are functional. A defect on either chromosome generally renders the transcription (simple copying) or translation (the active manufacture of gene product) of the genic subroutine nonfunctional. In such circumstances, no product is produced by the defect. However, the presence of such a defect is often tolerable to a diploid (redundantly encoded) phenotype. Gene product manufacture operates under an "on-demand," tightly-regulated active feedback loop, where demand controls source production. A single functional copy may suffice for all conditions other than extreme physiological stress. If only one working copy proves to be sufficient, the defect is said to be *recessive*.

But when two defective copies are present (and they need not be identical defects), complex physiologies are generally brought to a halt. It is this simple pattern of 3/4, 1/4 expression frequency that is characteristic of Mendelian genetics. The easy error lies in the misinterpretation of the 1/4 expression frequency.

"Dwarfism" is a common trait in domesticated plants and animals. In plants, dwarfism results from the unexpected absence of giberellic acids ([28] pp. 580–595). Although the trait is observed to often occur in classical Mendelian ratios, no

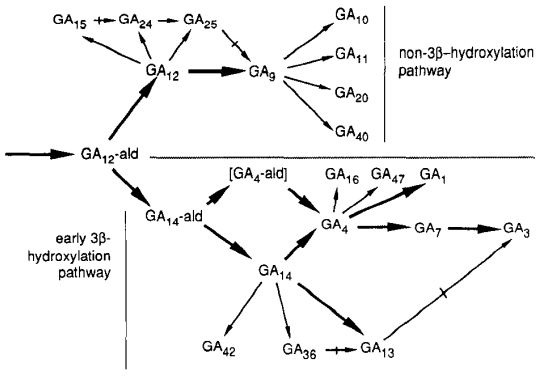

Fig. 6. A giberellic acid biosynthetic pathway following the synthesis of GA_{12}-aldehyde. giberellic acids are small molecule metabolites (phytohormones). In contrast, enzymes (not pictured) are complex catalytic molecules that are the posttranslationally modified products of gene translations. A bold arrow represents a primary pathway, a light arrow a minor pathway. Each arrow implies the action of one or more enzymes (after [28]).

actual "gene" for dwarfism exists. Nor do "genes" specifically exist for giberellic acids. giberellic acids are small-molecule metabolites, phytohormones, that are the manufactured products of multi-enzyme circuits (Fig. 6). A homozygously-expressed recessive defect in any enzyme anywhere within the giberellic acid biosynthetic pathway may completely disrupt giberellic acid production. Dwarfism, like most defects, is a syndrome. Although the causal defect may always be traced a specific point coding error, an innumerable number of similar point errors will create approximately the same physiological deficiency.

The great error is to label the defect as a "gene" for dwarfism. Although such usage remains unfortunately common, and can be marginally defended if used only as a verbal shorthand, it is a fundamental error that greatly misrepresents the informational physics at hand. Neither dwarfism nor the defective code is the selected quality. Rather, it is the presence of a functional molecule (most normally, a protein) produced by an operational genetic subroutine that was the quality built by iterative selection.

All code is intrinsically valueless in isolation; code only assumes value when it is translated into functional behavior. But the "fitness" value of a defect can only be measured by the degree of harm it does its bearer. It is a negative process and is not representative of the value functioning code imbues to the species, nor can it be a measure of the tolerance the species exhibits to allelic variation in the genetic subroutine.

Optimization is not perfection. Optimization is a "bubblegum-and-baling-wire" affair in which no better alternative exists. Optimization proceeds by sorting through the available behavioral variants. The result is a genome that is often extensively integrated for no reason more complex than that existing code is reused and reoptimized for a multitude of purposes. But the primary driving force underlying the integration of the genome is that the phenotype is judged as a unit working whole. Its value is not and cannot be measured piecemeal.

Among the very first to clearly argue these points was the Russian geneticist, Chetverikov. He emphatically rejected "the former notion of the mosaic structure of the organism

consisting of various independent genes. Each inherited trait is determined by not just some one gene but by their whole aggregate" ([29] p. 190, cited in [5] p. 558). This argument has been repeated many times since by Wright, Mayr, Dobzhansky, Stebbins, and Ayala, among many.

And yet as soon as the classical equation for mean genetic fitness, $\bar{w}$,

$$\bar{w} = p_{wAA}^2 + 2pq_{wAa} + q_{waa}^2 \tag{8}$$

for a single-locus, two-allele system is written on paper, a proper understanding of the nature of extensive gene product interactivity is put at risk. Equation (8) is explicitly correct only when A represents the set of all functional alleles and a an informational defect. An error, by its nature, is informationally trivial and isolated from the remainder of the genome. A defect is subject to selection in a highly directed manner that no complex trait can be. Any other philosophical extrapolation of the equation must be taken with great care.

B. Error 2: The Construction of an Entire Philosophy on a Mistaken Postulate

If the nature of the "gene" is misrepresented, a misrepresentation of the whole of the evolutionary process is unavoidable. Such a misinterpretation is evident in the following text:

"Honey bees suffer from an infectious disease called foul brood. This attacks the grubs in their cells. Of the domestic breeds used by beekeepers, some are more at risk from foul brood than others, and it turns out the difference between strains is, at least in some cases, a behavioural one. There are so-called hygenic strains that quickly stamp out epidemics by locating infected grubs, pulling them from their cells and throwing them out of the hive. The susceptible strains are susceptible because they do not practise this hygenic infanticide.... Rothenbuhler surmised that there might be two separate genes, one gene for *uncapping*, and one gene for *throwing out* [italics added].... This story illustrates a number of important points.... It shows that it can be perfectly proper to speak of 'a gene for behaviour so-and-so'" ([30] p. 64–66).

The author's conclusion profoundly misinterprets the nature of genes. As in all complex machineries, an infinity of failures may bring the machine to a halt. The point failure of any single cog, sprocket, or spring may be sufficient to cause the catastrophic failure of the entire machine. But the inverse is clearly not true. No single sprocket, cog, or spring can generate complex behaviors as an isolated component.

A point defect has been clearly confused with a complex behavior. While the mistake may initially seem extraordinary in its transparancy, it is an unfortunately common error. This simple mistake has been a persistent error that has permeated much of the history of mathematical genetics. But the real cost of the error accrues in its theoretical extrapolations. Such a fundamental misinterpretation of mechanism leads to a philosophy of evolutionary design that quickly becomes preposterous, "selfish" genetics.

Five thousand to 50 000 actively translated gene products are characteristic of metazoan genome sizes. On average, 10–50 % of the active genome appears to be translated in any one cell type. Constituent code representing basal cellular metabolism (commonly called *housekeeping* code) appears to represent 50–75 % of the code translated within all cells (10 000–20 000 gene functions in Deuterostomes, [31]). Much of this basal constituent code is presumed to be not characteristic of the species, but of the phylum, or higher, indicative of the antiquity of its origin. Tissue-specific code (another 10 000 to 20 000 gene functions in mammals and birds; often called *luxury* code) is evolved hierarchically on top of the basal genetic platform. The construction of all complex, well-conserved structures (an eye, a heart, or stereotypical behaviors) result from the expression, directly or indirectly, of virtually all of the translated genome. To speak of a "gene" for a complex behavior such as altruism, monogamy, or "uncapping" is simply nonsense and cannot be justified even as a verbal shorthand. It is, at its core, completely misleading.

C. Error 3: The Easy Reversal of Cause of Effect

Two simple but unfortunately common confusions currently plague much of biological theory, and to a degree, by consequence, evolutionary algorithm research. They are: 1) the proposition of selection *for* a specific trait in contrast to the *culling* of the least appropriate variants, and 2) the philosophical emphasis on genetic detail, from the bottom–up, rather than the processes that promote phenotypic optimization, from the top–down.

The question of whether selection selects *for* a trait or *culls* the least fit variants degenerates into simple semantics if the coding segments are informationally independent. If so, the process would be akin to drawing red and black balls from an urn. Selection for the red balls would be identical in process to selection culling the black balls.

But, therein lies the easy error. Even a statement of what may seem to be simple biology may be extrapolated into two completely disparate philosophies of evolutionary mechanism. A current example is dependent on the following often-quoted text: "Sexual selection depends on the success of certain individuals over others of the same sex" [22]. This simple sentence offers a significant opportunity for an easy reversal of cause and effect. The text may be read as selection for the most attractive males or as a mechanism of selectively culling the least healthy males.

Should sexual selection act to differentially select the most trait-attractive males [32], then a "Fisherian runaway" process of constant amplification of the attractive trait cannot be philosophically avoided. For whatever slight attraction exists, there will be persistent selective pressure to continuously exaggerate the trait, in isolation of the remainder of the genome, generation after generation, if its bearers are to continue to enjoy differential reproductive success. Moreover, there will be significant advantage in "counterfeiting" the trait (that is, decoupling the attractive trait from any intrinsic measure of the vigor or worth of the individual) so that its bearers may differentially reproduce, selfishly. Under such informational physics, "we must expect [the evolution of] lies and deceit" ([30] p. 70).

The alternative explanation is altogether different, both in cause and effect. Rather than selection "for" specific attractive traits, males bearing congential defects are exposed through prolonged demonstrations of vigor and behaviorally excluded from the breeding population. Much evidence exists that males are a relatively physiologically fragile, auxiliary sexual caste. Males, in the majority of animal species, live shorter lives, are more heavily parasitized, are more likely to succumb to starvation, trauma and stress, are less well genetically buffered, are inherently more strategically sacrificial than females in times of populational stress, and are often actively discriminated against during such periods. The prolonged and elaborate demonstrations of competitive vigor and pugnacity characteristic of males act as an effective genetic filter. This "honest advertisement" [33] of vigor insures that congenital defects present in the current population are not persistently reintroduced into the germline [33]–[35].

The conclusions the two philosophies imply are wholly at odds, as are their informational physics. The differences between the two informational physics are as stark as the differences between truth and deceit. In one, evolutionary advantage lies in maximized reproduction, through deceit if need be; in the other, advantage lies in the accurate detection and exclusion of defect bearers from the breeding population. In the first interpretation, benefit accrues only to the individual or his genes. The individual acts only in his own best interests, even when such acts are to the detriment of the group as a whole. In the second, benefit accrues solely to the evolving phyletic line. Individual organisms, like individual cells and tissue, can be sacrificed if sufficient informational benefit is obtained.

The first interpretation subtly restates two fundamental errors: 1) it promotes the evolution of coding structures which act to mediate, and promote their own survival probabilities, and 2) it restates the credit-assignment problem associated with the apportionment of worth to individual components or code structures in isolation of the system context in which they were developed. What value can be placed on any subroutine or component outside of the context in which it is used? Indeed, what value can be placed on a single component in context?

Highly polyparametric optimizations are the norm of nature. While it is now possible to decipher the genetics that codes for the construction of any specific mammalian hair type, it remains beyond current capabilities to assign any single fitness value to the deciphered code. Although the chemical structure of hair is relatively simple, the genetics that encodes that structure cannot be evaluated on the basis of any single criterion. The evolution of the protein hair rope is governed by a multitude of simultaneous criteria: the capacity to act as 1) a thermoregulatory blanket, 2) as a mode of crypsis, 3) as protection from insects and mites, 4) surface armoring, 5) mechanoreceptive sensory perception, 6) antibacterial and antifungal defense, 7) pheromone dispersion, 8) water repellency, as well as 9) a component of specific and individual recognition. Because each of these attributes is tightly coupled extrinsically to the various selection criteria of a constellation of other behaviors, a change in any singular genetic attribute must, by necessity, imply ramifications that impact variously throughout the evolving genome. Pleiotropy is an inescapable consequence of polyparametric optimization. No important, stabilized feature of the phenotype can likely be non-pleiotropic.

The fitness value that any isolated component brings to a system design is, in general, not equal to the harm it may induce should it fail. An o-ring gasket is a simple component, and in most systems tolerant of a wide range of variation in design for which no differences in the overall applicability or fitness of the system can be ascribed, but the failure of a single gasket in the space shuttle *Challenger* was fatal.

Evolutionary optimization integrates the entirety of a complex design, not merely by constantly readjusting and relinking the web of interactions for the purposes of generating increasingly appropriate behavior, but also—if not primarily—by minimizing the probabilities, costs, and consequences of the failures of individual components.

In any population of machines composed of a complexly constructed web of interactive effects, the increasingly selective exclusion of the least fit and least fault-resilient operates to increasingly integrate the whole of the lineage's behavior. Selection for individual components in highly pleiotropic systems in isolation of their effects on the whole becomes virtually impossible.

D. Error 4: The Celebration of Components Rather than Process

The fourth error recapitulates the third but is often expressed differently. Two approaches to the analysis of complex informational structures are possible: either from the bottom–up or from the top–down. Bottom–up analyses emphasize individual components and their interactions, disintegrating the evident complexity so that it may analyzed piecemeal. Top–down analyses emphasize extrinsically imposed forces and their physics, to the extent that they might be determined. Neither analytical approach can be completely satisfactory because both simplify nature. But neither are they equal in value, nor do they portend similar philosophies. Bottom–up analyses have inherent pitfalls. Where even very few components interact, there exists strong philosophical pressure to speak of "emergent properties," describing patterns of behavior not immediately predictable from the behaviors of the isolated individual components. The necessity to resort to emergent properties is a hallmark of any explanatory hypothesis that tends to reverse the actual flow of evolutionary causation.

Whenever emergent properties are introduced into a philosophy of evolutionary design, a higher-order mysteriousness is simultaneously introduced into the process that treads dangerously close to vitalism. Predictability and, most especially, inevitability disappear from the process. Evolution, from this perspective of randomly assembled components, proceeds by the inexplicable fits and starts of lucky happenstance.

This view mistates evolutionary causation and tends to make a tautology of the process. While random variation is the driving force of evolution, selection is the sole editor of that variation. The survival of the contending phenotypes is mediated wholly by their various positions on the fitness

landscape. The constant, random reshuffling of system components is assumed in Darwinian evolutionary theory. Indeed, the reshuffling is inescapable in a hot Boltzmannian universe. But selection too is inescapable in a finite arena and those variants that function better than others are differentially retained. This persistant selection moves a population of Malthusian trials inexorably, almost deterministically, towards the points of minimum system error on the adaptive topography.

Nor is the chain of causation silently evolved; causation leaves its stamp on the evolved structures. Should the physics of selection act in a manner such that individual coding structures are optimized, from the bottom–up, the resulting genetic basement must then inevitably become a collection of perfected "genes," informationally disconnected from one another, each operating to maximize its own optimal survival. But should the physics of selection operate only to minimize total system behavioral error, never directly acting on the coding structure itself, the result must be wholly reversed. The evolved phenotype must become composed of highly integrated "organs of extreme perfection" [21] while the genetic basement will inevitably become an overlain mixture of co-opted, duplicated, and highly interdependent code. It is only this second pattern that is found in nature.

The celebration of specific detail of a gene, a neuron, or a behavior, in isolation of its context, is a fundamental error. It has been an unfortunately common error, the result of which is virtually always a misrepresentation of not only the fundamental physics of the process of evolution, but also of what is being evolved.

E. Error 5: The Uncritical Emulation of Symptoms

The final error is the most subtle form of engineering error, simply because it is not always a mistake. But when it has proven to be an error, it has generally proven to be so severe that all future progress is shut off until the misguiding views are abandoned.

The mistake lies in too quickly emulating the symptoms of a particular process rather than pursuing a fundamental understanding of the underlying physics. The most overt symptom of naturally-evolved flight is the flapping of wings. But no practical, man-carrying ornithopter has ever been built. The secret to the physics of flight does not lie in the obvious flaps of the wing but in the far less obvious shape of the airfoil.

This form of symptomological error has been a persistent companion of artificial intelligence work. Indeed, Turing's classical test of intelligence emphasizes only the symptoms of intelligence as its measure, not the process itself, and the result has been that two generations of investigators have set off to model the symptoms of intelligence while expressing great reluctance to even define their goal.

A more restricted but ultimately perhaps as debilitating emulation of symptoms has recently become dogmatic of some simulated evolution work. Chromosomal inversion and especially crossing over have come to be advocated by the students of genetic algorithms to be essential and critical attributes of their specific evolutionary optimization technique [36]–[39].

Genetic algorithms are basically a proper simulation of Darwinian evolution. A population of trials is mutated and the best N are retained at each generation. The philosophical emphasis however is put on genetic mechanism, not on the process of phenotypic adaptation.

The greatest confusions in science and engineering have historically occurred when a process has been only slightly misrepresented. Success becomes its own validator of philosophical correctness. But that misrepresentation also becomes the stagnator of future work. No further progress can be made until a rectification in the understanding of the physics in force is regained.

Sexuality is a preeminent symptom of virtually all highly optimized, complex species. But if sexual recombination is initially presumed to be a necessary or highly beneficial feature of evolution, then all further work will tend to inevitably become philosophically constrained to model and mimic those specific attributes of sexual recombination that are considered to be fundamentally important. The question is: Is such a presumption, even when loosely stated, correct?

The absence of successful parthenogenetic lineages is *prima facie* evidence that the value of sexually mediated code recombination is pronounced. But the uncritical advocacy of a particular aspect of any phenomenon offers the possiblity of misinterpretation. Chromosomal recombination (sexual or allelic) is not a necessary constituent part of the evolutionary optimization process. Evolution would proceed with or without recombination. The evolutionary advantages that sex offers a species are twofold: 1) Sexually-mediated chromosomal recombination generates a very mild mechanism of increased mutagenesis that allows working variants of functional subroutines to be to be shuffled among phenotypic trials. This is a very subtle form of end-game optimization that dramatically shrinks the size of the optimization trial universe and thus greatly increases ($> 1000\times$) the rate of optimization to local conditions. 2) But the second advantage of sex may be more fundamental. Sexually-mediated chromosomal recombination operates to purge defects from the germline, defects that would otherwise rapidly degrade a parthenogenetic (asexual) lineage. The rate of mutational load on the species germline is not trivial. If organisms of any complexity are to be evolved, they cannot remain asexual beyond a certain complexity limit.

There exists a critical upper limit to the complexity level that may be evolved under a fixed (environmentally induced) mutational error rate. The governing inequality is

$$n\mu' > p \tag{9}$$

where n is the number of actively translated loci, μ' the basal thermodynamic error rate per locus, and p the ploidy (redundancy) number. Should $n\mu'$ much exceed p, error will accrue in the population faster than any possible level of selection can cull it from the germline. If $n\mu' >> p$, no individual progeny will exist without significant behavioral deviation from its parent. Reproduction must be very nearly replication if the ancient lineage of phylogenetically accrued knowledge is to be maintained. If that linkage is significantly broken at every replication, the evolutionary search will decay into a condition worse than enumeration. One of three choices

is presented to evolutionary design: 1) limit n, and thus limit complexity, 2) evolve mechanisms which reduce the effective mutational error rate, μ', or 3) greatly increase redundancy (polyploidy). Complexity above a prescribed limit cannot be evolved in the absence of the evolution of error suppression and recovery mechanisms.

The adoption of redundancy as an error suppression technique is an informational trap to the species. If at least one working copy of each critical gene is to be maintained, generation after generation, the redundancy count must grow to infinity at a rate greater than the number of generations experienced. However, as the redundancy count grows, the rate of evolution must concomitantly slow, due to simple numerical inertia. Indeed, as p approaches infinity, evolution ends. Genic behaviors that were "fixed-in" early in the sequence now swamp the effects of all novel mutations.

In sharp contrast to polyploid redundancy, sex expurgates defects from the germline through a redundancy-reduction step (meiosis). Even when primitively implemented, as in the pseudosexual nuclear cyclings of autogamous Protozoa, the effect is pronounced in its in reduction of accumulated mutational load from the germline [40]. In the majority of current genetic algorithm research, crossing over is taken to be equivalent to sex. That may, in fact, be basically correct (Appendix), but not for the reasons commonly stated. The invention of sexuality, for the two reasons described above, represents an extraordinary acceleration of the optimization process, and the advantages to a species that would adopt recombination are thus so great that its recurrent invention is assured.

Nonetheless, recombination is not functionally necessary to the evolutionary process. The philosophical error that potentially lies in any emulation is to so exaggerate a particular symptom to a point that the exaggeration replaces observed fact. It becomes the nature of such uncritical advocacy of a particular phenomenon to inherently promote a blindness in perspective that becomes difficult to later dispel. In that situation, the accurate determination of the relative worth of the various contributing processes becomes very difficult to assess.

IV. What Remains to be Accomplished

To date, the optimization problems addressed by the students of simulated evolution have been relatively simple. The majority of the problems have been combinatorial optimizations. Few have been the time-sequential, language-like, prediction-error minimizations that are far more characteristic of natural evolution. Although the use of the evolutionary algorithms have often demonstrated significant advantage over alternative optimization procedures, the simulations have remained nonetheless relatively sterile expressions of the complexity of naturally evolved informational structures.

There are several attributes of natural evolution—hierarchical organization, metamerism, epigenesis, and coloniality—that can be described as the great tricks of evolution but that have not yet been fully made part of the simulated evolutionary discussion. The incipient incorporation of these informational schemata into the eukaryotic cell lineage ca. 750–720 million years ago appears to be much of the impetus responsible for the explosion of diverse, complex, multicellular species at the beginning of the Cambrian [14].

The term *mere* in biology represents an irreducible unit or segment. *Metamerism* is the process by which a unit structure is duplicated a number of times, and in the process of duplication, often reoptimized for other purposes. The animals most normally associated with metameric constructions are the Annelida (segmented worms) and the Arthropoda (crustaceans, insects, spiders, etc.), but metamerism is a common theme throughout all of evolution.

Once the code exists to build a basic structure, it is reasonable to presume that it must be a relatively simple process to adapt it to other uses by endogenously duplicating subsections of the code, thereby generating the possibilities of multiple pathways of expression (epigenesis). In arthropods, segments are coded such that protopoditic code (that is, code encoding a basic leg structure) commands the construction of one pair of legs per segment. The code, however, is not executed in the same manner in every segment. The eight legs of a spider, the six legs of an insect, or the ten legs of a lobster are only identical laterally. They are differently manufactured as the segments are serially traversed. And in each segment, the functional purpose of the code is often altered. And the differences in function between segments can be startling. In one animal, the same generalized protopoditic code may encode the manufacture of sensory antennae, manipulative palps, chewing mandibles, grasping claws, mating cerci, or swimming caudal fins, in addition to legs.

The code in every segment is identical; indeed, but for minor exceptions, the code to build not only legs but the entire organism is present in every somatic cell of the individual. The metameric nature of the propoditic code is occasionally made clear when it fails to be expressed correctly. As in all complex systems, underlying mechanisms become most clearly transparent when the system partially fails to function. A well-known homeotic mutation in *Drosophila* is antennapaedia. The result of anntenapaedia is the incomplete and inappropriate manufacture of a leg extending from the fly's head where an antenna would normally appear.

Once metameric code is built and optimized, the question becomes principally one of regulation. Not only are the functional purposes of the segments often substantially altered as the chain of repeated segments progresses, their expression is sequenced through a hierarchy of regulatory steps. Polydactly is a common defect in terrestrial vertebrates (including humans) when regulation fails. One to many additional fingers are manufactured during embryogenesis. The excess digits are generally small, only partially functional, and taper off as they round the hand or foot.

Precision high-order regulation becomes functionally critical to any form of complexity evolved through metamerism. But higher-order regulation also provides significant opportunities for enhanced plasticity. Indeed, the informational nature of metamerism recapitulates much of the philosophy of object-oriented programming. In higher-order evolutionary sequences, it is the changing relative expression of a large col-

lection of "objects" that represents the majority of evolution, not substantial changes in the objects themselves. The easy modification of that relative expression is sequenced through a command hierarchy that allows for, if not outrightly promotes, rapid evolutionary modification, and this rapidity of system behavioral modification has been one of the more often touted advantages of object-oriented programming techniques.

Metamerism also inherently implies modularity, and thus additional evolutionary plasticity. Modularity permits *mosaic evolution,* in which one part of the body plan is modified more rapidly than others. Once distinct code exists to build fingers, the fingers may be independently—and more easily—modified to become the wings of bats or pterosaurs, or welded together to become feet and hooves, or freed from locomotion so that they may manipulate tools, while other key organs, such as the eyes, already highly optimized for their specific tasks, may remain relatively stable.

Evolution intrinsically repeats informational patterns of value. Those patterns and procedures that possess fundamental advantage will be recurrently rediscovered, and the value of metamerism is not shallow. Metamerism is one of the tricks quickly learned by every junior programmer as the simplest and fastest way to build complexity into an encoded structure. And yet, there have been only a very few hints in the current work (e.g., [37], [58]) to suggest systems of simulated evolution that would autonomously promote the duplication and subsequent reoptimization of existing structures and code. Obtaining this result does not seem all that difficult, but it has not yet been achieved. Obviously, the models are incomplete. Or perhaps just insufficiently complex. If it is to be done correctly, it cannot be forced (programmed into the structure). The phenomenon of metamerism must evolve spontaneously. The challenge that remains is to properly identify those conditions that promote its evolution.

Nor are we seeing any sign of incipient colonality in the current work. The advantages of colonality do not generally lie in a great increase in the complexity of behavioral repertoire of a social species over that characteristic of solitary species, as may be initially believed, but lie in the significant increase in reliability, redundancy, and most especially, the parallel expression of a myriad of behaviors simultaneously [26].

Undoubtedly, the same advantageous physics that drives the evolution of multicellularity similarly drives the evolution of sociality. The individual cell is the only metabolic engine of life; all other higher-order organizations are complex colonial constructions of these cells. Higher-order organizations have evolved and are stabilized only through the evolution of extensive intercellular communication links that promote the cooperation and behavioral synchronization of the individual cells. When taken as a ratio, the number of cell-internal signals to cell-external signals decreases by ca. 100:1. The same approximate decrease holds for the number of intercellular signals when compared to the number of signals between individual members of a colonial society. It may be that no higher level of organization is possible simply because of the progressive diminishment of signal densities.

The evolution of colonality and metamerism are intimately interwoven phenomena. Cooperative colonality evolves in only those situations where individual phenotypes of a single species begin to interact in a mutually advantageous manner. For true colonality to evolve, contextually-induced (epigenetic) metamerism must be present, as so must near-genetic identicality, in all of the members of the colony. All of the species' code must exist as an integrated "bundle" in every phenotype, but expressed in a manner such that only a relatively small proportion of the code is actually engaged in any one phenotypic morph. It is highly probable that this is the only path that promotes the evolution of stable colonality and hierarchically distinct "objects" such as organs, organisms, and societies.

The incorporation of these higher-level processes into the emerging engineering discussion is important if the evolutionary simulations are to be applied to anything other than simple optimizations. Behavior is the sole quality directly optimized by evolution. Each of these informational mechanisms—hierarchy, metamerism, epigenesis, and coloniality—not only greatly increases behavioral plasticity but the range of applicable problems as well.

The form of machine intelligence that may ultimately arise from evolutionary simulations will almost certainly be similar to human intelligence in its functions, but it may not be particularly anthropomorphic in its implementations. Intelligence has been built into natural processes in three ways: phylogenetically, sociogenetically, or ontogenetically. Although quite differently accomplished, they are functionally equivalent processes. Each represents a mechanism of retaining appropriate behaviors learned through trial-and-error experience.

Although the analogy must be drawn with caution, from the point of view of the evolving species, extrinsically, sex and intelligence act as if they were basically equivalent processes. Both are processes learned phylogenetically. And both processes accelerate the optimization of the lineage by offering the adopting species much greater behavioral plasticity, thus significantly enhancing its competitive advantage. Recombination and imagination may similarly be argued to be somewhat equivalent processes. Each seems to promote the rapid reshuffling of variants drawn from a discrete pool of variants, and in the doing, creates new trial combinations. The variants drawn, however, are not random mutations. Under normal circumstances, each variant has demonstrated some survival value in the past.

There currently exists only one human species, and its lineage has been characterized by a relatively monophyletic line of descent. Humans exist among a relatively small group of sister species. There are only seven to ten species of great apes (depending upon preferred taxonomic divisions) now in existence. All other mammals comprise only 4300 species; birds add but another 8700 species. But in great contrast, insects have evolved into perhaps 7 000 000 to 80 000 000 distinct species [53].

The extraordinary differences in species counts between the single human species and the insects speak directly to the manner by which adaptive intelligence has been enacted in the two groups. Human intelligence has evolved to become a general-purpose platform on which appropriate behavior may be learned without further engaging any form of physiological

or morphological modification. Insects, on the other hand, are essentially behaviorally non-plastic. However, the behavioral repertoire of an insect should not be minimized; it is more complex, more elaborate, and more subtle than any piece of electronic machinery now built. But the repertoire is also almost wholly hard-coded, derived directly from germline DNA, and further appropriate modifications of the species' behavior require modifications to this code.

The adaptive plasticity of a species of insect does not lie in the individual, as it does in humans. It lies in the rapid evolution of the lineage, which can be exceedingly plastic. This plasticity is due to several factors: 1) rather shallow developmental pathways that appear to be easily co-opted, 2) enormous population sizes, and 3) relatively short individual lifetimes. And both groups, insects and primates, have independently discovered the competitive advantages of sociality: fault-tolerance, resiliency, parallelism, co-operative care, greater flexibility in learning, and an independence from the fate of individuals. The most social insect species are more intensely social (in that the roles of their castes are more exaggerated, more communicative, and more interdependent) than any mammalian species.

In natural circumstances, the active modification of the code in a phylogenetically evolving lineage eventually progresses to a point that precludes any further sexual communication with its progenitor lineage, for reasons perhaps no more complicated than simple mechanical incompatibilities in the code. At that point, the modified population has become a distinct species. But the same isolating conditions that promote non-communicating speciation do not necessarily need to be part of a high-speed, artificially evolving process of machine intelligence.

Specializations in behavior derived through sexual recombination and ontogenetic intelligence would appear to represent two completely different pathways toward the high-speed evolution of subtly appropriate behavior. But for all the apparent differences between these two approaches, they are profoundly convergent in their capacities to accelerate the evolution of appropriate behavior. For this reason alone, they are functionally equivalent learning processes that may not be all that readily distinguishable when implemented in machine form, especially when the co-operative task partitioning intrinsic to epigenetic metamerism becomes part of the evolutionary algorithm.

Indeed, the differences in the two pathways to intelligence are illusory. The individual human is composed of ca. 6×10^{13} cells, but there is no single cell where intelligence resides. Those patterns of behavior that would commonly be described as intelligence exist because of the co-operative interdependence of a great mass of rather identical units (cells) organized into distinct castes of behavior (tissue). The capacity for intelligent behavior does not "emerge" from this mass. It is selected for its advantageous properties, as a unit whole, and is evolved throughout the lineage of trials, phylogenetically. Intelligence is not the end-product of evolution, nor is it a distinct process. The evolution of appropriate system behavior permeates every aspect of the process of evolution.

V. Appendix

A. The Biological Role and Function of Crossing Over

Because of the current emphasis placed on crossing over within genetic algorithm research, a review of the biological function and purpose of crossing over, as it is currently understood, is warranted. In distinct contrast to inter-organismal chromosomal recombination (sex), crossing over is a cell-internal intranuclear process that appears to be under active promotion by the processes that regulate meiotic nuclear division.

If every gene were encoded as an independent chromosome, crossing over would be unneccessary. But, in mammals, ca. 4000 gene functions reside on a single chromosome. Otherwise fit alleles potentially suffer the deleterious consequences of being physically linked with a severe defect resident anywhere on a chromosome. Crossing over would seem to offer the possibility of breaking such linkage disequilibria, informationally separating fit alleles from the accidental consequences of physically residing near a defect.

The phenomenon of crossing over was discovered during the period 1908–1915 by Thomas Hunt Morgan and his students. The discovery that genes could cross between chromosomes came as a great shock, similar to the excitement Barbara McClintock's discovery of transposable elements ("jumping genes") created during the 1970s. "As Muller [48] has characterized it...'the fact that Morgan's evidence for [genes] crossing over [from one chromosome to the other] and his suggestion that genes further apart cross over more frequently was a thunderclap, hardly second to the discovery of Mendelism'" ([5] p. 753). In the 80 years since, however, the value and role of crossing over has been steadily devalued from its earlier presumed importance as a fundamental evolutionary process.

Crossing over exists, but it is not necessary to the process of meiosis (the redundancy-reduction step characteristic of cellular gametogenesis in sexual species). Indeed, in *Drosophila* males, no potential for crossing over exists because no chiasmata form ([27] pp. 146–154, [42]). (*Chiasma* are the visible cytological events where two arms of homologous chromatids overlap.)

In the males of a great many species, the rates of *allelic recombination* (synonymous with an early definition of crossing over) is either much reduced or altogether absent. In Lepidoptera (butterflies, moths, skippers) however, the situation is reversed, as was first discovered by Sturtevant in 1915. He found that female silkworms and wax moths do not undergo allelic recombination because there is no formation of chiasmata, whereas males do form chiasmata. In Diptera (flies), males are heterogametic; they possess one pair of unpaired (non-homologous) sex chromosomes. In contrast, in *Lepidoptera,* females are heterogametic. What informational advantage this sex-linked suppression of chiasma formation confers to the species is not clear, but the reduction in the formation of chiasmata in the heterogametic sex is probably not coincidental, but rather "programmed."

Neither is the advantage that crossing over, when it exists, offers the species clear. Two recent contending hypotheses

are those of Maynard Smith [43] and Bernstein, Hopf, and Michod [44]. Maynard Smith argues a role and purpose for crossover not unlike sex itself. In this view, crossing over acts as a mutagenesis accelerator by increasing the rate of allelic recombination, in effect, a kind of sex on top of sex. In contrast, Bernstein *et al.*, who argue a DNA repair role for crossing over, also argue that the rates of crossing over are insufficient to maintain the accelerator of adapation that Maynard Smith proposes. "The fraction of physical recombination events [the formation of visible chiasmata] that result in allelic recombination is infinitesimally small....The fraction of total recombination events that result in [actual] allelic exchange at meiosis is...less than 2.6×10^{-6}" [44].

These points can be agreed upon: 1) chiasmata form during meiosis and, in some species, during mitosis, 2) the rates of chiasmata formation are not synonymous with the rates of allelic recombination, 3) the process almost certainly has some—but currently unclear—evolutionary purpose.

The principal argument supporting the last statement is the general recognition that crossing over is a "promoted" process rather than an uncorrected, persistent expression of residual error. The evidence for the active intranuclear promotion of crossing over is two-fold: 1) Proteins with strand exchange activity have now been identified in organisms as diverse as the bacteriophage T4 (a virus), bacteria, lower eukaryotes, and human cells. This commonality implies that DNA strand exchange is a property of a ubiquitous class of proteins that can be referred to as *recombinases* ([45] and references therein). And, 2) the crossing-over process must be presumed to be under active promotion because it can be so easily suppressed in one sex or the other. Only an actively coded process can be easily suppressed. A random error process cannot likely be subject to easy sex-specific suppression. If it were, and if the process had no positive evolutionary value, it would quickly disappear in both sexes.

It is however also possible to argue with some lesser force of logic that current evidence suggests no evolutionary role for crossing over. It is simply a cytological process that results in no significant genomic alterations. In human females, the rate of chiasma formation is 1.89 per bivalent. If the rate of allelic recombination is as low as Bernstein *et al.* calculate, the process is insignificant in its effects when compared to sexually-mediated chromosomal recombination. DNA cannot be broken at any arbitrary point. The mechanism that allows true allelic recombination to occur at all is perhaps no more complicated than that the majority of the DNA on a chromosome is pseudogenetic and no longer actively translated. Breaking such structure is quite likely to be often informationally neutral, and thus tolerable.

Among the more intriguing aspects of crossing over is that it is almost always differentially expressed in the two sexes, and in that, shares broad characteristics with a number of other genetical phenomena. *Haplodiploidy* is a sex-differentiated phenomenon that differentially exposes defects in the male genome to direct selection. Under haplodiploidy, males are haploid and thus practical allelic recombination during meiosis is functionally impossible. *Parahaploidy* is a directly related condition that has been found to exist in some mites. In this condition, male and female zygotes are fully diploid, but the male sheds his paternal genome sometime prior to sexual maturation, and thus allelic recombination in the male is again impossible. In all placental mammals, the rate of chiasma formation during meiosis is ca. 30 % higher in the female than the male. Achiasmatic meiosis is the rule in most insects that are male-heterogametic. The sum of these phenomena suggests that crossing over serves an informational maintenance purpose rather than acting as the accelerator of adaptation that Maynard Smith has suggested.

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