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13. ABSTRACT (Maximum 200 words)

Self-determined systems are usually studied by researchers with unrelated terminology and few known common principles. This workshop was aimed at bringing together scientists whose research directly confronts the problems of analyzing, explaining and building self-determined systems. We hoped that sharing their results and interpretations at the meeting would inspire cross pollination of ideas from different viewpoints and lead to a more unified approach and language to understanding self-determination. The workshop format and discussions were aimed at discovering underlying principles while amplifying little known links between scientific fields. The emphasis was on discovering tools and mechanisms that have general application to research problems in biology, neuroscience, psychology and computer science. Fifteen researchers came together to explore these issues at Harvard University, January 6-9, 1994. Together, they brought their expertise from Biology, Neuroscience, Developmental Psychology and Computational Modeling.

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Workshop on Self-Determination in Developing and Evolving Systems: Final Report

February 18, 1994

Symbus Technology
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Workshop on Self-Determination in Developing and Evolving Systems

Summary

The concept of "self" is at the heart of what it means to be alive. As obvious as the self feels to us is as mysterious it is to understand. Clearly the self develops in a life cycle and evolves across life cycles, but where does it comes from? Clearly the self can only be understood in relation to "other" or "group" or "environment", but are the roles of these relationships? The self requires energy and resources to sustain itself through transactions with its world, but what drives those transactions? We have defined self-determination as the ability of a contained process to regulate and direct its perpetuation and growth. Self-perpetuation here is viewed as both surviving during a life cycle and reproducing across life cycles. Growth in self-determination is not simply an unfolding of a maturation process. The main issue of growth in both development and evolution is innovation. Where does innovation come from and how does it lead to increasing the spatial and temporal scope of relationships and transactions with the self?

We can talk about the self at different levels of life from single cells to brains to human individuals to cultural institutions. It is appealing and in some sense, satisfying to find a continuity of principles and mechanisms across all levels of life. This then, is the prime motive for organizing this workshop. It was geared to exploring how specific principles of self-determination can be applied across all levels of life.

Self-determined systems are usually studied by researchers with unrelated terminology and few known common principles. This workshop was aimed at bringing together scientists whose research directly confronts the problems of analyzing, explaining and building self-determined systems. We hoped that sharing their results and interpretations at the meeting would inspire cross pollination of ideas from different viewpoints and lead to a more unified approach and language to understanding self-determination. The workshop format and discussions were aimed at discovering underlying principles while amplifying little known links between scientific fields. The emphasis was on discovering tools and mechanisms that have general application to research problems in biology, neuroscience, psychology and computer science.

The workshop had two main practical challenges. Could we talk to each other in a common language and could we agree on anything concrete. By focusing on natural examples and engineering design problems, we hoped to minimize the tendency for discussions to get bogged down and become overly theoretical, abstract and vague.

Fifteen researchers came together to explore these issues at Harvard University, January 6-9, 1994. Together, they brought their expertise from Biology, Neuroscience, Developmental Psychology and Computational Modeling. As the meeting progressed a number of critical questions and themes emerged and crystallized. What is the self and what is its purpose? What are the sources of innovation in development and evolution? How do hierarchies emerge in life? How can relationships and transactions from one level of life be mapped to another? The enclosed abstracts and outlines present how the participants viewed these questions.

Workshop on Self-Determination in Developing and Evolving Systems 2 of 2

Saturday, January 8

7:30 am to 8:30 am	breakfast
8:30 am to 9:20 am	**Dr. Richard Ryan** - University of Rochester - Organizational Principles in Human Behavior and Psychological Development: Analysis and Application to Motivation and Volition
9:20 am to 10:10 am	**Dr. Edward Deci** - University of Rochester - Effects of the Social Contexts on Self-Determination
10:10 am to 10:20 am	coffee break
10:20 am to 11:10 am	**Dr. Thomas Ray** - ATR HIP Japan- Evolution in other universes
11:10 am to noon	**Dr. David Ackley** - Bellcore Towards the Evolution of Communication
12:15 to 1:30 pm	Lunch
1:30 pm to 2:20 pm	**Dr. Jay Mittenthal** - University of Illinois Level-invariant processes of self-determination: Scenario, examples and implications
2:20 pm to 3:10 pm	**Dr. Michael Kuperstein** - Symbus Technology - Single Cell Agents to Multi-Cell Hierarchies: Constraints, Mechanisms and Simulations
3:10 pm to 3:20pm	coffee break
3:20 pm to 4:10 pm	**Dr. Domenico Parisi** - CNR Institute of Psychology- Rome, Italy Artificial Life and the Study of Behavioral Change
6:30 pm to 7:30 pm	Dinner
7:30 pm to 8:10 pm	Discussion led by **Dr. Edward Deci**
8:15 pm to 9:00 pm	Discussion led by **Dr. William Wimsatt**

Sunday, January 9

7:30 am to 8:30 am	breakfast
8:30 am to 9:15 am	Discussion led by **Dr. James Shapiro**
9:20 am to 10:10 am	Discussion led by **Dr. Domenico Parisi**
10:10 am to 10:20 am	coffee break
10:20 am to 11:00 am	Discussion led by **Dr Terry Deacon**
11:00 am to 11:50 am	Discussion led by **Dr. Michael Kuperstein**
11:50 am to noon	Closing Remarks

Additional Workshop Attendees

Funding Agencies
Dr. Terry Allard, ONR Program Manager

Press
Harry Stanton, Bradford Book, MIT Press
Mitchell Waldrop, Science
Roger Lewin, American Scientist and New Scientist
John Rennie, Scientific American
George Johnson, New York Times

Workshop Topic Questions

1. Are there any shared principles of self-determination between evolution and development across different scales of living systems?

2. Is Evolution directional?

   a. Can evolution be directional but not progressive?

   b. If cells were to direct their evolution like people direct their development, then the genes would need some form of environmental feedback. Are there any plausible mechanisms for such feedback?

3. What is the balance between stability and plasticity?

   a. How do living systems enable growth which is inherently unstable while maintaining stable performance?

   b. How are conflicting environmental cues transcended into higher-level syntheses?

   c. How can a process create a structure which modifies its process in novel directions while surviving novel incompatibilities?

   d. What are the energetics of material and/or information processes that allow the development of increasingly more ordered structures?

4. What is the balance between differentiation and integration?

   a. How can these systems differentiate their response to improve selectivity while integrating their response to improve generalization?

   b. How can these systems differentiate their response to optimize division of labor while integrating their response to optimize group function?

5. What is the balance between neighboring interactions and hierarchical interactions?

   a. What mechanisms balance competitive and cooperative interactions in symbiosis?

   b. What mechanisms balance lower order and higher order systems in an hierarchy?

6. What are the causes of new variability in development and evolution?

   a. For the generation of evolutionary innovation, what is the contribution of random mutations, directed mutation, gene conversion, symbiogenesis, fusion, jumping genes or other mechanisms?

   b. For the generation of developmental innovation, what is the contribution of performance errors, exploration or other mechanisms?

Workshop on Self-Determination in Developing and Evolving Systems 1 of 2

Thursday, January 6

6:00 pm to 7:00 pm	Welcoming and Introductory Remarks - **Dr. Michael Kuperstein** and **Dr. Terry Deacon**
7:00 pm to 8:00 pm	Dinner
8:00 pm to 9:00 pm	Opening talk - **Dr. Stephen J. Gould** - Harvard University
9:00 pm to 10:00 pm	Social Hour and beverages

Friday, January 7

7:30 am to 8:30 am	breakfast
8:30 am to 9:20 am	**Dr. James Shapiro** - University of Chicago Genome Organization and Reorganization
9:20 am to 10:10 am	**Dr. Frank Ruddle** - Yale University Evolution of the Homeobox Gene Family
10:10 am to 10:20 am	coffee break
10:20 am to 11:10 am	**Dr. William Wimsatt** - University of Chicago The Evolution of Generative Structures
11:10 am to noon	**Dr. Terry Deacon** - McLean Hospital, Harvard University- The significance of displacement of selection, the role of non-genetic information in brain development and the uniqueness of human brains
12:15 to 1:30 pm	Lunch
1:30 pm to 2:20 pm	**Dr. William Calvin** - University of Washington - Cerebral Darwinian Processes on the Time-scale of Thought and Action
2:20 pm to 3:10 pm	**Dr. Jason Brown** - NYU Medical Center, Dept of Neurology Microgenesis
3:10 pm to 3:20pm	coffee break
3:20 pm to 4:10 pm	**Dr. Peter Corning** - Institute for the Study of Complex Systems- Synergy, Symbiosis and Self-Organization in the Evolution of Complex Systems
6:30 pm to 7:30 pm	Dinner
7:30 pm to 8:10 pm	Discussion led by **Dr. Frank Ruddle**
8:15 pm to 9:00 pm	Discussion led by **Dr. Peter Corning**

Genome organization and reorganization

by James A. Shapiro, Department of
Biochemistry and Molecular Biology, University of Chicago, 920 E. 58th St, Chicago, Ill. 60637 (312-702-1625, jsha@midway.uchicago.edu)

ABSTRACT Like the revolution brought about by quantum physics, the discoveries of molecular genetics have led to major changes in our understanding of heredity. While classical genetics was concerned with the mechanics of inheritance in cell lineages, molecular genetics has focussed on the dynamic aspects of genome function. Molecular techniques have deconstructed classical genetic elements, such as the gene, into intricate mosaics composed of many distinct genetic motifs, and we have learned about the existence of multiple overlapping and interacting genetic codes. Studies of cell and developmental biology have revealed the existence of hierarchically integrated genomic systems involving distant regions of the genome. Comparisons of related genetic structures within and between species have produced a radically new picture of genomic evolution by reorganization of genetic motifs to create new system architectures. Studies of the mechanisms of DNA change have revealed a wide variety of *natural genetic engineering* systems resident in diverse cell types. These systems can effect many different kinds of DNA reoganizations, sometimes involving many sites within the genome within a single cell generation. Like all other complex biochemical functions, natural genetic engineering systems are subject to cellular control regimes. Examination of a contemporary case of evolutionary change, the emergence of bacterial antibiotic resistance in response to antimicrobial chemotherapy, illustrates how genomes evolve by the addition and rearrangement of discrete DNA elements.

Shapiro, J.A. 1991. Genomes as smart systems. Genetica 84, 3-4.
Shapiro, J.A. 1992. Natural genetic engineering in evolution. Genetica 86, 99-111.

Genome organization and reorganization

by James A. Shapiro

(1) Three basic messages:

- o Genomes are composed of a large number of different types of coding elements.
- o These coding elements are organized logically and hierarchically in various combinations within the genome. The architecture of this organization facilitates the participation of DNA in the mechanics of hereditary transmission and in cellular information processing.
- o Cells possess multiple natural genetic engineering systems which permit the rapid reorganization of coding elements. These complex biochemical systems are subject to cellular regulatory regimes.

(2) Different types of coding elements (selected):

2a) Protein coding segments
- 2a1) domains rather than entire proteins systems)
- 2a2) exons ==> possibility of differential splicing

2b) Transcription signals
- 2b1) deconstruction of lac operon (operators, promoters)
- 2b2) logical organization of binding sites (lambda imm region)
  - undetectable failure rate or repression
  - multivalent nature of transcription factors (negative or positive depending upon context)
  - cooperativity at multiple levels
  - efficiency based on logical structure, not thermodynamic constants
  - release of repression by elimination of protein cooperativity (RecA as genome sensor/regulator)

2c) Repetitive DNA
- 2c1) Factor binding sites (transcription, replication, segregation, recombination, etc)
- 2c2) centromeric, telomeric repeats (alphoid DNA -chromosome mechanics, spatial organization of the genome)
- 2c3) Other satellites, heterochromatin (position effects on gene expression; taxonomic specificity)

2c4) Dispersed repetitive elements (SINES, LINES, taxonomic specificity)
2c5) Specific genome architectures - Britton & Davidson concept

(3) Natural genetic engineering systems

3a) in vitro genetic engineering based on enzymes extracted from cells
3b) Mobile genetic elements (TIBS cartoon)
3b1) Episomes
3b2) DNA-based transposable elements (multiple possible rearrangements)
3b3) Retroviruses and retrotransposons (> 1 class)
3c) Biological control of mobile element activity
3c1) Mu-dependent coding sequence fusions
3c2) Hybrid dysgenesis (premeiotic bursts of activity ==> clusters of gametes)
3d) Developmental DNA rearrangements
3d1) Immune system
3d2) ciliated protozoa (massive genome rearrangements in one cell generation)
3e) Role of natural genetic engineering in evolution of bacterial antibiotic resistance

(4) Conclusions:

o Genomes are complex, hierarchically organized information storage and retrieval systems composed of many different classes of coding modules
o Genome architecture (i.e. the organization of these different modules) is more important than the properties of individual molecular interactions in controlling the process of information retrieval from DNA
o Genetic change involves the action of natural genetic engineering systems and, consequently, is a highly regulated cell biological process rather than a matter of chemistry and physics

Evolution of the Homeobox Gene Family

by Frank Ruddle, Yale University

Abstract:

Homeotic genes in the mouse have been a productive system of experimental analysis. It has been possible to show that these genes play an important role in pattern formation, lineage commitment, and differentiation.

The mammalian (human and mouse) homeobox genes are organized as four gene clusters on four different chromosomes. There are 38 Hox genes in all with 11 genes in cluster Hoxa and 9 genes each in clusters Hoxb-d. DNA sequence analysis shows that the serial order of genes is the same between clusters, suggesting their origin by cluster duplication. The serial order of genes in the clusters is also colinear with the HOM-C in Drosophila, and this has led to the postulation of an ancestral cluster with five homeobox genes. The postulate is supported by the identification of cluster related genes in a number of extant primitive forms. In addition to the homeobox cluster genes there are more divergent, non-clustered Hox genes that map to scattered sites in the mammalian genomes. These 'Dhox" genes number in excess of fifty and continue to be accessed at a high rate. Representatives of all the major phyletic groups possess both Hox and Dhox genes, including sponges and cnidarians. As a broad generalization, the Hox cluster genes appear to be primarily involved in pattern formation along the anterior/posterior axis, whereas the Dhox genes contribute more to a variety of "downstream" events such as lineage commitment and differentiation.

Homeobox Genes and Evolution

Frank Ruddle, Yale University

A. Homeotic Genes
    1. Gene structure
    2. Function as transcription factors
    3. Regulatory aspects of gene duplication
B. Structural Organization of Hox Gene Clusters
    1. Expansion of clusters by gene duplication
    2. Multiplication of clusters by genome duplication
    3. Paralogous or cognate homeobox genes
    4. Structural arguments for homology
C. Conservation of Function within the Hox Gene Clusters
    1. Paralogous relationships among linked genes
    2. Collinearity between clusters and the anterior/posterior axis
    3. Gene swapping experiments
D. Derived Homeobox (Dhox) Genes
    1. Distribution in the genome
    2. Relationlships based on nucleotide sequence
    3. Functional attributes
E. PCR Based Survey Methodology for Homeobox Sequences
F. Homeobox Sequences in Primitive Forms
    1. Sponges
    2. Cnidarians
    3. Planarians
    4. Caenorhabditis
G. Homeobox Sequences in Protosomes
    1. Molluscs
    2. Annelids
    3. Arthropods
H. Homeobox Sequences in Deuterostomes
    1. Echinoderms
    2. Ascidians
    3. Acorn worm
    4. Amphioxus
    5. Lamprey
I. Conclusions
    1. Functional organization of the genome
    2. Relationships between Hox and Dhox genes
    3. Nature of ancestral metazoans
    4. Hox genes and punctuated equilibrium
    5. Animal metazoans as monophyletic

The Evolution of Generative Structures

lecture outline

W. C. Wimsatt 12-15-93

Conference on Self-Determination in Developing and Evolving Systems
Cambridge, MA., January 5-9, 1994

1. Introduction: Short comments on the relation of generative entrenchment to standard population genetic approaches to modelling evolution [see handout #1]

2. Historical commentary: a lineage in pictures (a series of slides)

a. von Baer's developmental sequence
b. Darwin's phylogenies.
c. Haeckel: from phylogenies to cellular descent trees
d. from Weismann to Weismannism and the elaboration of cellular descent trees
e. from cellular descent trees to generative entrenchment

[Note that this lineage can be viewed either as a historical commentary on a theory, or itself as a lineage of generative structures, this time in the cognitive realm of the evolution of scientific theories, rather than of biological evolution. I will return to this ambiguity or multiplicity of interpretations near the end of the talk, though you might observe the multiplicity of columns and parallels between them in handout #3, "parallels betwen biological, cognitive and scientific evolution".]

3. The developmental lock and generative entrenchment [see handout #2]

a. Simon's complex lock.
b. Simon's simple or near decomposeable lock.
(comment on application to reductionistic heuristics.)
c. The developmental lock.
d. simple generalizations: the 100 position lock and k; the multiparameter lock.

4. The DL as a model of development

A. **Assumptions:**

a. wheels as stages of development, left to right from earlier to later.
b. changes in wheel position at stage i as mutations expressed at stage i.
c. sequential dependence of combinations as product of causal dependence of features at later stages on features at earlier stages.
d. adaptive problems for phenotype assumed to be independent at different stages, but success requires getting adaptive solutions at all stages.

B. **Some simple consequences:**

e. probability of successful trial at stage m wheels from right is k^m; so there is an exponentially declining probability of getting a mutation which is adaptive as expression moves earlier in development.
f. earlier features tend to have more things depending on them than later features. Thus:
1. the probability of earlier changes being adaptive is lower.
2. earlier changes will tend to have more pervasive and major effects.
(the net effect of 1 and 2 together is that increasing proportions of earlier changes will have more strongly negative effects.)
g. define **generative entrenchment** of of a trait, process, behavior or structure **x** as how many things depend on **x**. (a variety of different measures of GE are possible)
h. then earlier features tend to be more generatively entrenched than later features.
i. from f above, earlier developmental stages should be increasingly evolutionarily conservative.
j. this explains von Baer's law: Differentiation proceeds from the general to the particular, where generality can be interpreted taxonomically, morphologically, or functionally.
k. earlier (or more generatively entrenched) things should tend to be older (but not for science!)
l. things that stay around longer should get increasingly resistant to change (by becoming generatively entrenched through the acquisition of "downstream modifiers").
m. deeply entrenched features in smaller structures should change more rapidly than deeply

entrenched features in larger ones. (note argument for the adaptiveness of near-decomposability here, since ND or causal modularity decreases GE.)

n. evolution is irreversible.

o. phylogenetic and contemporary patterns of stasis, change, and covariation should be usable to infer relative generative entrenchment.

5. A series of more realistic models of systems showing generative entrenchment: [limitations of model expressed in parentheses]

1. Simple developmental locks and extensions of them (producing a single serially dependent structure having the logical organization of a tree.) Wimsatt, 1981, 1984, 1986. [but no parallel subsystems; everything at a given serial stage is in the same subsystem]

2. Cellular descent trees and their causal isomorphs, with interactions mediated only by descent. (Branching tree structures of causal interactions) (Arthur, 1984, 1988). [allows parallel subsystems, but presumes causal monophyly or downstream causal divergence]

3. Series-parallel networks of developmental locks. (Rasmussen, 1987). [as above, but downstream causal hybridization possible]

4. Causal reaction structures given by digraphs--arbitrarily connected sets of nodes having no internal gating structure. Scope of entrnechment defined in terms of reachability. (indicates topology of connection only). (e.g., Kauffman, 1985; Schank and Wimsatt, 1987, Wimsatt and Schank, 1988) [arbitrarily complex and unconstrained causal topologies].

5. Networks of Boolean automata: like (4), but nodes are Boolean functions. (Kauffman, 1969 for gene control networks; McCulloch and Pitts, 1943 for neural networks.) [allows arbitrarily complex combinatorial logic of tasks and interactions in a discrete state system]

6. A selection of consequences from some of the more advanced models:

a. Rasmussen, JTB, 1987 analyzed developmental mutants of *Drosophila* to produce a deficiency based flow-chart of the developmental program of *Drosophila* using generative entrenchment a year before a basically identical map was published (in *Nature*, 1988) using more standard arguments, illustrating the power of comparative data and a GE model for analyzing the structure of developmental programs.

b. More realistic models of the evolution of gene control networks using GE as a factor affecting fitness confirmed some of Kauffman's (1985) results, while correcting others. In particular, it proved possible to maintain significantly larger proportions of an array of selected alleles (and all of the more important ones) in the face of mutation and the effects of genetic load than with Kauffman's results with digraph models. These results suggested the importance of modelling large genomes with heterogeneous selection coefficients. (Schank and Wimsatt, 1988).

c. Models of the evolution of complex adaptations suggested that changes in the architecture of developmental programs over extended periods of evolutionary time could inflate the number of genes that could be maintained by selection by 2 to 5 orders of magnitude by conversion of "hard" selection processes to "soft" selection processes. (Wimsatt and Schank, 1988) [A parallel process for scientific theories acts to allow the maintainence and evolution of larger theoretical structures by focussing disagreements leading to modifications on a small fraction of the possible foci for change at any given time--even though anything and everything may be "up for grabs" at one time or another.]

d. Simulations with large supposedly additive multi-locus haploid truncation selection-mutation equilibrium models suggested by GE constraints confirmed the results in (b) above, but further revealed new heretofore undiscovered emergent phenomena. These phenomena appear in any models in which mean Darwinian fitness can undergo significant change (which means any selection models with a large number of loci), and in which the loci do not all have identical selection coefficients[1]: (1) a new kind of "frequency-dependent" selection mediated by changes in mean Darwinian fitness, which results in (2) a strange sort of "inter-locus" compensation, in which decreases in frequency at loci with small to negligible selection coefficients will act to increase the frequency of important (strongly selected) alleles to near-fixation. This suggests a mechanism which could "lock in" traits with higher GE even when mutations in them are not unconditional lethals, and "unlock" them under

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[1]Strange as it may seem, apparently, no quantitative genetic selection models with this set of assumptions have been studied prior to ours!

conditions which lead to transitory increases in mean Darwinian fitness--e.g., such as major decreases in competition due to mass extinction. [Handouts # 4,5]

7. Consequences for cognitive development: [See handout #3 for 7, 8 and 9]

a. Parallel phenomena to a number of those indicated above should hold for cognitive development. Not only does generative entrenchment provide the basis for a re-analysis of the innate-acquired distinction along different lines which eliminate many of the paradoxes of the classical distinction, but it captures a broader range of "innate" phenomena than any prior analysis, and does so in a way which explains the relationships among the different criteria offered for innateness--relationships which have gone explained, or simply had to be assumed on prior analyses. (Wimsatt, 1986).

b. Similar parallels should hold for the differentiation and elaboration of cognitive structures through learning and on a developmental time-scale.

8. Consequences for Scientific Evolution and theories of Cultural Change:

A very large number of parallels exist for the role of generative entrenchment in explaining differential rates of change in the evolution of scientific theories, and the character of our heuristics for making such changes as conservatively as possible. The parallels include prediction of a declining frequency of scientific revolutions of increasing size, and the "hardening" of core assumptions of paradigms with time in ways that give generalizeable and robust alternative explanations for many of the features Thomas Kuhn found in the history of science. These include explanations for the analytic-synthetic distinction, the conversion of empirical results into quasi-definitional or "syntactic" constraints through use, the advantages of limiting-case and inter-level reductions, the modularity of theories and models, an explanation for the criteria for evaluating "good' definitions, and an identification and analysis of factors which allow scientific and cultural evolution to take place much faster than biological evolution.

9. Picturing Weismannism--A Case-Study of Scientific Evolution:

The lineage of pictures used to begin the historical commentary provide readily distinguishable lineages of 'cultural organisms' whose evolution we can trace, and in which we can find evidence of some of the phenomena discussed above. (see Griesemer and Wimsatt, 1988). Diagrams of Weismannism (which some of these cell-lineage diagrams became) underwent an enormous adaptive radiation as neo-Darwinism grew in influence, and had many features commonly associated with biological phylogenies. In 110 diagrams spanning the last 100 years, we have found so far: Descent with and without modification, adaptive radiation, with "bottom-heavy" clades, specialization for specific problems or conceptual niches, extinction, local adaptive survival, and continued survival (?) though maladapted to the new problem context. We found clear evidence that these diagrams were not epiphenomenal to the evolution of the scientific theories, but played an important role in mediating workers' understanding of them--including crucial *mis*understandings of Weismann's views which have supported an overly reductionistic interpretation of evolutionary theory--following Williams and Dawkins--by most modern writers. We have also found (paralleling the move from empirical to analytic truth of widely used features) the standardization and schematization of Weismann diagrams, until they are even used for diagrams which *attack* Weismann's theory. More pictures are shown to illustrate these points.

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HO# 1

The Relation of Population Genetic and Generative Entrenchment approaches to modelling Evolution: W. C. Wimsatt 8-7-93

I. Standard population genetics models: {populations are described in terms of frequencies, numbers, and sometimes both for different purposes}

stage 1: **Population(t_0)--[demography, mating rules (+sexual selection)]-->mating pairs**

stage 2: **--[Mendelian genetics (+cytology, molecular, gametic selection)]-->offspring genotypes**

stage 3: **--[(development, physiology, ecology)->(phenotype, selection)]-->Population(t_1)**

•In this, the phenotypes (which actually enter in at each stage, but are treated as if they enter in at only the 3rd stage) are treated as black boxes which are dummy variables for the processes of development and selection, and are used to assign fitnesses as scalar multipliers of gene or genotype frequencies. Adaptations enter in only through their effects on changing gene frequencies. The simplest models omit the 1st stage by folding it into the 3rd stage and drastically simplify the 2nd and 3rd stages.

•Population genetics basically gets its power through the combination of Mendelian genetics, applied to the description of populations, with fitness assignments either assumed or derived from other sources.

•For future use below, note that no matter how complicated the model, the Δq equation (for change in gene frequency) MUST always have $\underline{W}$ in the denominator, since the relative numbers of genotypes after selection are divided by $\underline{W}$ to get the (normalized) genotypic frequencies required for stages 1 and 2.

II. Generative Entrenchment models take an abstract description of the causal structure of the developing phenotype or of the phenotype/environment interaction to assess how changes in the developmental program will affect fitness, and thereby to assign fitnesses to different genotypes.

•It thus can be used to flesh out the black boxes at all stages of the standard model above, (particularly in development) giving reasons for assignments of particular distributions of fitnesses to different genotypes, and explanations for adaptations and for the stability of some non-adaptive phenotypic features.

•As such, it has lead to the construction of population genetic models of a purely conventional type suggested by the above which have led to novel conclusions (such as the existence of $\underline{W}$-mediated frequency-dependent selection and inter-locus compensation, and another look at the differences between hard and soft selection.)

•Since some features of causal networks are generic (in the sense of Kauffman), these both identify features which we don't require selectionist explanations for, and thereby also identify "soft constraints" which should play an important role in evolutionary models. (e.g., differential generative entrenchment).

•Since entrenched features should tend to be evolutionarily conservative, and we have independent criteria for judging the entrenchment of a feature relative to other features (temporal order, causal dependence, breadth of effects), we can use comparative phylogenetic and developmental information to get information about the structure of developmental programs.

•The emphasis on generators, rather than genes has lead to some interesting conceptual innovations--e.g., the recognition that features of the environment as well as genes can have an important generative role, a reconceptualization of the innate-acquired, and of the *a priori-a posteriori* and analytic-synthetic distinctions, and that generative models may be useful in some cases where genetic-based evolutionary models are difficult to apply (e.g., for cultural evolution where we have better access to developmental dependencies than in biology, but where the rules for mating and inheritance are much messier.)

•The use of information about development allows rather direct modelling of intuitions not readily captured on standard population genetic models--e.g., the explanation of parallels between ontogeny and phylogeny, the importance of "Baldwin effect" mechanisms in evolution, and the role of critical periods and canalization in development.

•The GE model allows generation of results not accessible on another approach--e.g., the expectable increase in size of the number of genes which can be maintained by selection over evolutionary time of Wimsatt and Schank (1988).

•As with the use of Hardy-Weinberg equilibrium, the failure of GE models can be an important diagnostic tool for seeing which of its assumptions are violated. Thus, evolutionary lability of early developmental stages (contra expectations) points to a limited number of mechanisms for avoiding the constraints of the model, which can arise sometimes in parallel, sometimes differently in biological and in cultural evolution.

Figure 9-1: The Developmental Lock

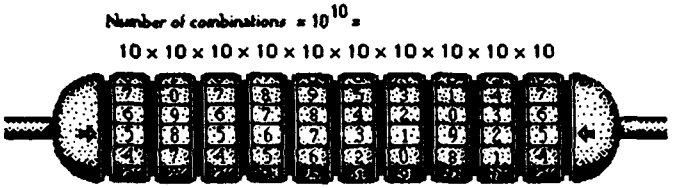


Figure 9-1a. Simon's "complex" lock--10 wheels with 10 positions per wheel. In the "complex" lock, the correct combination is only discoverable as a complete solution. (No clues are given for partial solutions).
Expected number of trials = $10^{10}/2 = 5 \times 10^9$.

Figure 9-1b. Simon's "simple" lock--just as above, but a faint click is heard when each wheel is turned to its correct position, allowing independent solutions to parts of the combination. (The advantage of near-decomposeability in problem solutions is the ratio of the expected number of trials for the two locks = $5 \times 10^9 / 50 = 10^8$.)

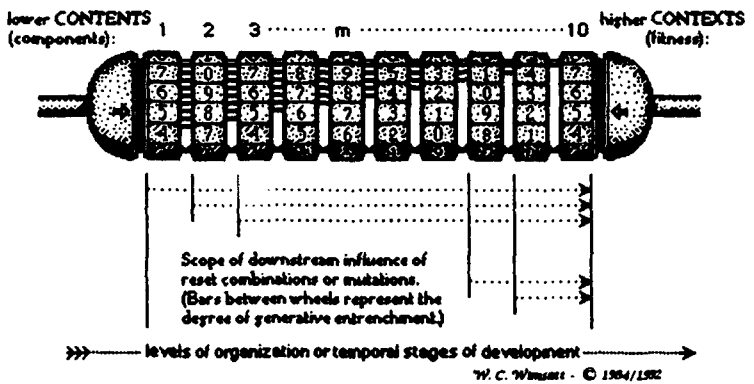


Figure 9-1c. The Developmental Lock: This lock is a hybrid of Simon's 2 locks. Suppose a "click" is heard when each wheel is set to its (conditionally) correct position, but what position is correct is a function of the actual positions (whether correct or not) of any wheels to the left of it, so that a change in position of any wheel randomly resets the combinations of all wheels to the right of it. (Simple if worked from left to right, (since the partial solutions to the left are not disturbed by work on wheels to the right) but complex if worked from right to left (in the sense that partial solutions are not preserved).

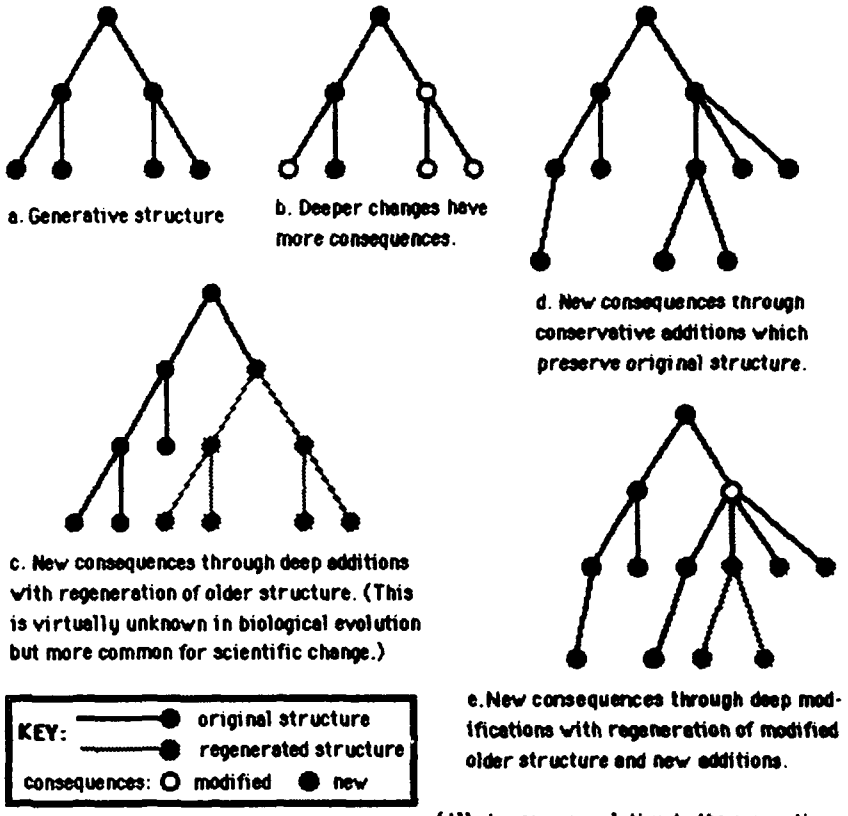


Figure 2: Types of Change in Generative Structures

(All changes are relative to the generative structure given in a, except for e, which is a modification of the structure given in d.)

Increasingly complex and realistic models of systems showing generative entrenchment:

1. Simple developmental locks and extensions of them (producing a single serially dependent structure having the logical organization of a tree.) Wimsatt, 1986.
2. Cellular descent trees and their causal isomorphs, with interactions mediated only by descent. (Branching tree structures of causal interactions) (Arth[illegible]94, 1988).
3. Series-parallel networks of developmental locks [illegible]en, 1987).
4. Causal reaction structures given by digraphs--[illegible]y connected sets of nodes having no internal gating structure. (indicates topology of connection only). (e.g., Kauffman, 1985; Schank and Wimsatt, 1987, Wimsatt and Schank, 1988)
5. Networks of Boolean automata: like (4), but nodes are Boolean functions. (Kauffman, 1969 for gene control networks; McCulloch and Pitts, 1943 for neural networks.)

-W. C. Wimsatt 12-14-93

HO#2

Ho*3

General Consequences of the Developmental Lock or Generative Entrenchment:

W. C. Wimsatt 8-26-86 (latest rev. 12-16-93)

Items in bold indicate significant differences between biological or cognitive and scientific development; see also evidential status.

Evidential status: **X**, *x* = analytical, definitional result; X = empirically grounded; *X*, *X* = weak empirical, conjectural, ? = status unknown (• = subject to Raff-style counterexamples.) (these symbols may also be used in combination for mixed or ambiguous cases.)

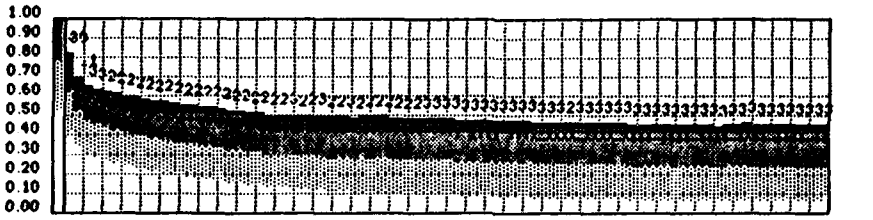
X's in developmental, evolutionary columns indicates locus of phenomenon; Arrow(s)indicates causal direction.

	BIOLOGICAL		COGNITIVE		SCIENTIFIC		
	devel	evol	devel	evol	devel	evol	Remarks:
1. Earlier features have higher probability of being required for later ones •	X		X		X,?	?	(from model) (and observation?)
2. Earlier features tend to have higher # of "downstream" dependents •	X		*x*,*X*?			X	(from model) (indirectly from 7,8)
3. exponential decline of adaptive mutations with earliness in development •	X		X		X		(from model) (qualitatively confirmed)
5. von Baer's law: Differentiation proceeds from general to particular•	X		X		X,?	?	
7,8. early/deep anomalies have greater effect•	X		X		*x*		(observable, and from #2)
12. # of modifiable sites increase at later developmental stages	*X*?		*X*?		X		(Arthur; Q-ed by Raff)
13d. Parallels in structure of diverse internal systems is evidence for GE of structure	*X*		*X*		*X*		(examples anyone?)
17. Differentiation increases GE	X		X		X		
18. Controlled processes GE control elements	X		X		X		
??Rasmussen: Correlation of growth more restricted for later acting mutations	X		*X*?		*x*		(on Raff's view also)
4a. Conservativeness of evolution at early developmental stages •		X		X		X	
4b. Most evolution occurs at later stages		X		X		X	
11. (Callebaut): evolution is irreversible		X		X		*X*?	(argument from #10)
6. earlier features are older •	X——>X		X——>X		no		
9. Simpler structure--> faster evolution	X——>X		X---->X		X——>X		
14a. High GE structures acquire dependent adaptations more rapidly (assuming breadth)	X——>X		X---->X		X——>X		
26. Evol of releasing stimuli w/o changes in adaptive structure of behavior	X——>X		X——>X		X——>X		
10. things around longer become GE'd	X<——X		X<——X		X<——X		
13. comparative info reveals GE structure	X<----X		X<----X		X<----X		(but see Mabee, 1993)
14b.Highly, stably selected, low GE structures should increase GE	X<——X		X<----X		X<——X		(argument from #10)
16/29. Selection incr. reliability, canalization of high GE structures, and conversely via #10	X<——X		X<----X		X<——X		(argument from #10)
19. Selection incr. portability of GE elements	HOX mutants?		?		X<——X		
20. Evol. of complex adaptations requires differential selection intensities for parts	X<----X		X<----X		X<----X		
21. Differential degrees of GE are generic properties of selected systems arising spontaneously thru symmetry-breaking transforms	X<----X		X<----X		X<----X		
22. GE, protection of elements from modification allows generation and maintainence of larger structures	X<——X		X<----X		X<——X		(cf. Campbell's "doubt/trust ratio" and hard vs soft selection)
30. Differential rates of evolution , sex---> selection for quasi-independence	X<——X		?		X<——X		prediction:Is this true?
25. Quasi-independence allows "piecemeal engineering", adaptive modification of parts, and more rapid evolution (reverse effect too!)	X<=====>X		?		X<=====>X		recombination analog (see Holland's GA's)

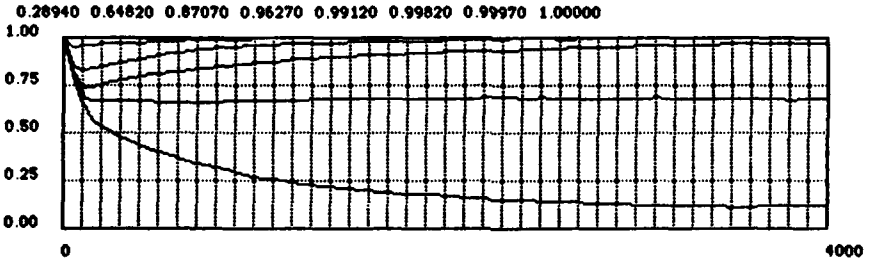
Possible means for modification of deep elements in biology: (1) Selection for quasi-independence decreases interaction of elements of a sub-system with other parts of the system; selection for presence or absence in different environments makes centralized control desireable; then selection for alternative states of a GE element becomes possible. (2) Maternal effect gene is changed to earlier acting embryo gene, with unchanged effect on embryo. (3) Other changes eliminate need for early feature (e.g., for feeding in sea urchin pluteï because of larger eggs). (4) heterochrony in quasi-independent developmental subsystems (this is a name, not an explanation). (5) functional neutrality (possibly designed, through selection for canalized states). (6) "Baldwin effect" or "genetic assimilation" mechanisms involving change of releasing mechanism under selection--w/o change of released behavior. (7) Raff's "complexity catastrophe" and global --> local transition. (8) Improbable "hopeful" monsters--basically, a bail-out of last resort.

Possible means for deep modifications in conceptual evolution: (1) The relatively low cost of production of conceptual variants, making it possible to try a larger number of them.-(in part because many of the variants are not followed up--they are "early developmental lethals"--see #24). (2) Unlike the situation in biology, in conceptual change, we do not have to *adopt* variants we are considering, allowing us to consider variants with lower fitness if they have "sufficient promise." (3) Although a theory is a coadapted structure, we can ignore that and decompose it, working on its parts in isolation and then recombining them to *get an acceptable solution. (Pseudo-near-decomposeability)* (4) The ability to localize faults in a theoretical network (cf. Glymour, (1980) contra the 'Quine-Duhem thesis') allows identification of the component which needs changing and direction of all of the efforts to produce variants to where it can do the most good. (This is one kind of "non-randomness" in the production of cultural variation not found for the production of biological variation.) (5) The ability to use the direction of error to indicate the direction of corrective modification increases the proportional yield of adaptive variants. (This is a second kind of "non-randomness.") (6) *"Baldwin effect" analog changes range or area of application* of (usually formal) theory--usually regarded as ampliative, not deep (exc. in new area?) (7) reaxiomatizing to make a derived result fundamental, or incorporating a result taken from another theory and used early in generation of given theory as intrinsic to the theory which uses it. (Like maternal effect->embryonic gene) (8) designed functional neutrality allowing intersubstitutability from a class of deep assumptions (often to customize a theory for diverse applications).

50 Run Average of gene-frequency trajectories in a 248 locus haploid model with genes in 5 fitness classes: note frequency reversals in top 3 trajectories.



0.26940 0.64820 0.87070 0.96270 0.99120 0.99820 0.99970 1.00000



GeneNets2.12 *random Seeds:*

Genes = 100 1: 1094000000 2: 1052000000 3: 665400000 4: 1934000000 5: 1383000000 6: 1862000000 7: 305200000

Connections = 248 8: 1834000000 9: 1999000000 10: 1410000000 11: 1090000000 12: 1975000000 13: 1621000000 14: 466900(

Pop. N = 10 15: 1816000000 16: 204900000 17: 1557000000 18: 1362000000 19: 1206000000 20: 1110000000 21: 19010(

Mut. Rate = 0.0025000 22: 1770000000 23: 459900000 24: 1727000000 25: 680500000 26: 832500000 27: 1003000000 28: 1769000(

Sel. Intensity = 1.00 29: 1822000000 30: 1019000000 31: 997200000 32: 955400000 33: 1099000000 34: 1803000000 35: 204400(

Base Fitness = 0.000 36: 827200000 37: 528600000 38: 1479000000 39: 281700000 40: 1772000000 41: 412200000 42: 13660000(

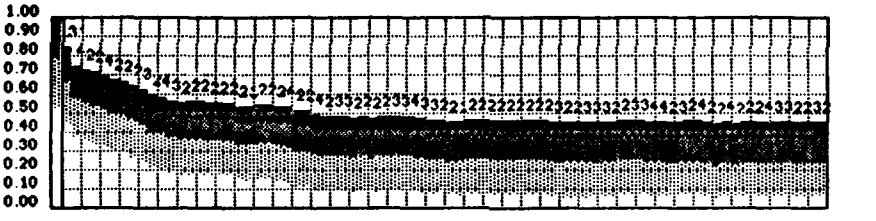
K = -1.00 43: 706300000 44: 1439000000 45: 573200000 46: 1884000000 47: 1892000000 48: 725700000 49: 1834000(

Run = 50 50: 493800000

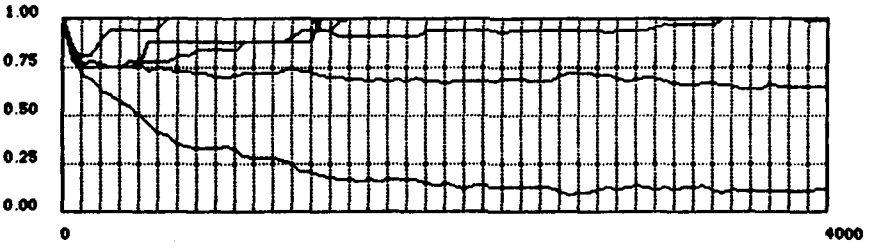
Fitness Types: 1: -0.1000 2: -0.0500 3: -0.0250 4: -0.0125 5: -0.0063 Run time: -717.06 Up time: -6912.89

Gene-frequency trajectories in Run # 50, showing stochastic deviations from average--deliberately induced by choice of a small population size (N=10).

W.C. Wimmer - 1988



0.26940 0.64820 0.87070 0.96270 0.99120 0.99820 0.99970 1.00000



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Connections = 248 8: 1834000000 9: 1999000000 10: 1410000000 11: 1090000000 12: 1975000000 13: 1621000000 14: 466900(

Pop. N = 10 15: 1816000000 16: 204900000 17: 1557000000 18: 1362000000 19: 1206000000 20: 1110000000 21: 19010(

Mut. Rate = 0.0025000 22: 1770000000 23: 459900000 24: 1727000000 25: 680500000 26: 832500000 27: 1003000000 28: 1769000(

Sel. Intensity = 1.00 29: 1822000000 30: 1019000000 31: 997200000 32: 955400000 33: 1099000000 34: 1803000000 35: 204400(

Base Fitness = 0.000 36: 827200000 37: 528600000 38: 1479000000 39: 281700000 40: 1772000000 41: 412200000 42: 13660000(

K = -1.00 43: 706300000 44: 1439000000 45: 573200000 46: 1884000000 47: 1892000000 48: 725700000 49: 1834000(

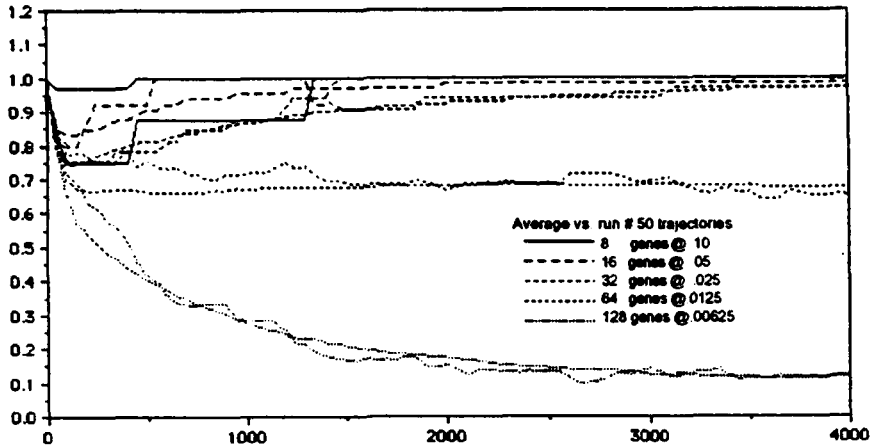
Run = 50 50: 493800000

Fitness Types: 1: -0.1000 2: -0.0500 3: -0.0250 4: -0.0125 5: -0.0063 Run time: -720.02 Up time: -6915.86

Inter-Locus Deviation Adjustments

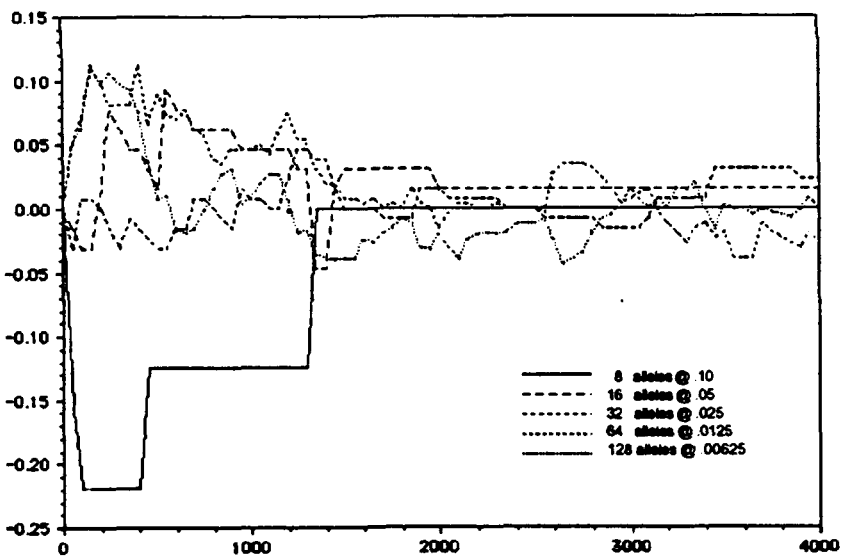
W. C. Wimsatt/J. C. Schank 5-19-93

EVOLUTION (FROM OPTIMUM) OF GENE FREQUENCIES IN A HETEROGENEOUS HAPLOID 248 LOCUS SYSTEM UNDER TRUNCATION SELECTION WITH SMALL (N=10) POPULATION SIZE



Frequency trajectories of genes in 5 fitness classes--Average (or equilibrium) trajectories (relatively smooth curves) vs. trajectories in an individual run (#50).

FIGURE 1: Note that the trajectories for the genes in the different fitness classes are quite different, and that , even though (as with the first class) there are sometimes quite large differences between the averaged and individual run trajectories initially, the differences disappear and appear to asymp- totically approach (or fluctuate around) the same value. Note also that while the deviations of the first class from its average value is negative and very large in between generations 0 and 1400, the devia- tions in the other 4 classes all seem to be positive in this same interval. This suggests a phenomenon of inter-class compensation which demands further analysis.

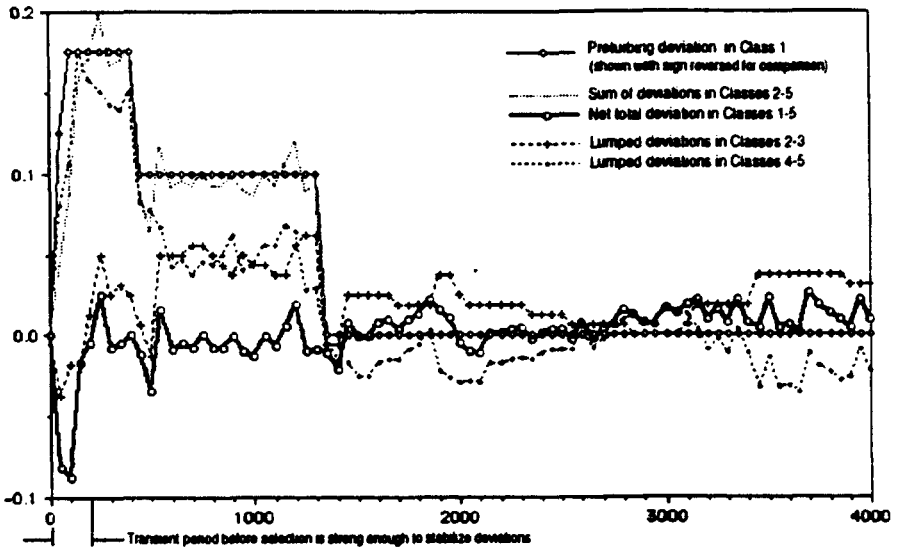


Deviations from Equilibrium Trajectories in Successive Generations

FIGURE 2: Deviations from 50-run averages in frequencies of various fitness classes of alleles, in run #50, which had, by chance, an unusually large deviation in frequency in the first fitness class (produced by loss due to drift of alleles at 2 out of 8 loci). Notice that the larger fitness classes (.1, .05, and .025) are obviously quantized in their frequencies after the first 200 generations (indicating fixation at all loci which can be changed only by back-mutation). Notice also that deviations in the 4 smaller classes appear to be positive during the period when the deviations in the first class are negative, indicating cross-class compensation to minimize net deviation.

HO #5

DEVIATION ADJUSTMENT IN VARIOUS FITNESS CLASSES FOR A PRIMARY PRETURBATION IN THE LARGEST FITNESS CLASS IN A 248 LOCUS HAPLOID TRUNCATION-SELECTION MODEL



Deviations in frequency of various allele fitness classes in run #50 from 50-run average in successive generations

FIGURE 3: In this run, 248 alleles in 5 fitness classes (.10, .05, .025, .0125, and .00625), in numbers of 8, 16, 32, 64, and 128 are subjected to mutation, and approach a selection-mutation equilibrium in which their frequencies are determined by their fitnesses, the mutation rate, and with fluctuations functions of population size. In this particular run, two out of eight of the fittest alleles were lost due to drift, and then reestablished by back-mutations after generations 400 and 1300. (Populations are sampled every 50 generations.) These primary fluctuations induced countervailing fluctuations in the opposite directions in all other fitness classes. The 50 run average is used to estimate the equilibrium tendencies of the system. The primary or preturbing fluctuations are shown with negative sign so that they can be compared directly with the sum of all other fluctuations (Sum 2-5) yielding a net deviation for all 5 classes (Sum 1-5) which is usually less than the deviations in any single class. Classes 2 and 3, and classes 4 and 5 are lumped together into 2 heterogeneous classes, with the latter lumped class having 4 times as many alleles, each with 1/4th of the fitness effect of the former. Notice that when the 1st class goes back to equilibrium, (at which point the 2-5 average merges with the 1-5 average) class 4+5 appears to counteract and adjust to the less frequent but more grossly quantized excursions of class 2+3. In each case, the lower- numbered classes are more subject to drift and loss of alleles due to fixation, making them less able to respond to net deviations in equilibrium. The response suggests a probabilistic analogue to a fourier decomposition, with the more frequent smaller fitness alleles giving more rapid and finer tuning, and the less frequent larger fitness classes giving larger but coarser responses. This case thus also turns out to be a nice illustration of a different sort of hierarchial near-decomposability, (sensu Simon, 1962), with different fitness classes responding on different time scales. The original losses occur within a 200 generation transient period, during which decreases in mean fitness inflate the relative fitness contributions of all alleles, leading to reversals in equilibrium frequency trajectories for alleles in the 3 highest fitness classes. Visible quantization appears in the top 3 fitness classes when drift losses keep frequencies below their equilibrium levels until back mutations occur (with different expected waiting times in the 3 classes).

–W. C. Wimsatt 1989

The significance of displacement of selection, the role of non-genetic information in brain development, and the uniqueness of human brains

Terrence W. Deacon, Boston University / Harvard Medical School

1. Scala naturae assumptions about brain function and brain evolution are still prominent and remain a major impediment to advancement of the field.
a. One classic assumption about how brains evolve is that they tend to increase in size and capability under broad selection for ever increasing intelligence; that evolution proceeds in a persistently progressive trend toward increased mental function.
b. Another common assumption is that there is progressive accretion of new structures and that newer structures subserve progressively higher functions.

2. These assumptions have become tacitly incorporated into functional theories.
a. The theory of mass effect (distributed processing) and numerical scaling of intelligence.
b. The theory of hierarchic processing in which top levels are last added and last activated.
c. Classic philosophical assumptions of associationism are simply recast in neuroanatomic and functional terms in most modern theories of brain function and are supported by fallacious evolutionary assumptions.

3. The two major evolutionary assumptions involved (size=processing power and progressive addition of higher-order new associative systems) are undermined by current neuroembryological data, and so their correlative functional theories are likely fallacious as well.
a. There is not a clear sense in which brain size correlates with intelligence.
b. A major problem of determining the consequences of differences in brain size is the non-isometry of neuronal and connectivity increases with larger size. Larger brains are of necessity organized differently than smaller brains. They have fewer connections are less integrated.
c. Developmental sequences do not recapitulate phylogenetic sequences of brain evolution. Late maturing systems are not necessarily newer, and early maturing systems are not necessarily older. The whole conception of what is newer or older needs revision.
d. It might be better to think of conserved versus modified developmental information.
e. Conceptions of what aspects of neuronal processing are *higher* or *lower* on a functional hierarchy or in terms of processing sequences cannot be derived from ideas of what structures are newer or older, which

are more primary or integrative, etc.

4. Neuroembryological constraints imply that:
a. Structures previously thought to be added serially in evolution are instead the result of species differences in differentiation of the same substrate that are determined by proportional differences between afferent axon populations and target neuron populations. These relationships are markedly affected by overall brain size and relationships between linked CNS and PNS systems.
b. Differences in relative sizes of structures do not necessarily indicate relative functional importance. Allometries (non-isometric scaling relationships) among brain structures in species of differing brain sizes reflect
c. Connectional specificity is initially highly degenerate and final synaptic domains are determined by competitive trophic processes.
d. Connectional architecture and corresponding functional differentiation are determined systemically not solely by local information.

5. How did the evolution of the nervous system lead to such an indirect morphogenetic process? The theory of masked/displaced selection.
a. Masked selection occurs when an extragenomic source redundantly provides the same information as some genetic source. If it is reliably present it will reduce selection pressure on the corresponding genetic sources and allow them to degenerate.
b. Prolonged (in evolutionary terms) masking will result in irreversible modification of the masked genes.
c. Subsequent removal of the external information source will be progressively more deleterious, the longer the organism has had to adapt to the presence of the external information.
d. This leads to displacement of selection for function from the original information source to others, because under the threat of loss of the external information source, selection will favor any other modifications that even indirectly help to maintain the presence of that source.
e. Though extragenomic masking offers the most dramatic cause of displacement of selection, it can also arise in a number of other circumstances, at most levels of organism-ecosystem *design*. These include internal masking by various processes of duplication (e.g. gene duplication, cell duplication, structural enlargement), responses to selfish DNA (e.g. suppression of *outlaw* effects, segregation distortion), selfish cells (e.g. reversion/transformation of cell lines), and endoparasites that impose a cost on the rest of the organism.

6. Displacement of selection leads to increased integration of functions and robustness of morphogenetic mechanisms.
a. Displaced selection disperses onto a wide variety of independent

genomic sources that need have only one incidental feature in common: they all affect the source of masking information.
b. Because multiple routes of causality are likely to evolve independently as a result of displacement, its effects can be additive or complementary or overdetermined.
c. Consequently, there will be a tendency to evolve more synergistic functional organization, and since each effect is to some extent redundant each will produce additional masking of each other, and lead to secondary and tertiary displacement effects, further distributing selection over a progressively wider range of loci.

7. Displacement of selection is a factor in the evolution of individuation and hierarchical organization.
a. This results in systems that give the appearance of underdetermination in embryogenesis because specific local relationships are not so often determined by specific locally expressed gene products but by a diversity of indirect and individually insufficient influences that just *happen* to converge at that time and place during development. However, the systemic determination that results is, in generally, both more highly buffered against noise and a more reliable determiner of target form, than any more direct mechanism would be.
b. The irreversible tendency to develop increased integration with respect to extrinsic information sources is a source for increasing self-determination and individuation.
c. Once individuation becomes entrenched at one level of organization, similar displacement processes acting with respect to groups of individuals at that level will have systematizing effects at the higher level of organization. Thus, the tendency for displacement effects to produce individuation also tends irreversibly to select for further hierarchical organization.

8. Examples in nervous system development/evolution/function.
a. Patterns of systemic determination in mammalian brain development are evident at all levels of development. Examples of underdetermination of cortical regional differentiation and afferent and efferent connections will be offered. Examples from heterotopic and cross-species neuronal grafting experiments demonstrate both the extent of non-specific distributed mechanisms and conservatism of brain architectonic processes.
b. Consequences for brain evolution are that evolutionary modification of either peripheral or central relationships produce systemic but highly buffered changes in CNS organization. The same developmental information can produce very different adult brain structure under different conditions. Changes in size can therefore produce emergent structural changes.

c. Examples of masked selection affecting nervous system development as a result of behavioral evolution include development of visual, locomotor and social-emotional functions. The effect of primate infant carrying on the evolution of emotional and locomotor behavior is particularly striking.
d. Human brain evolution and the evolution of symbolic reference provide an example of a fundamentally unique emergent function that did not require any *new* brain structures. It also shows how a behavioral adaptation can unmask a variety of neuronal systems by redistributing selection in novel ways. Symbolic reference processes themselves represent the emergence of a new level of top-down functional organization; a new level of self-determination that is to some considerable extent supra-organismic.
e. The complex multilevel structure of the adaptation for symbolic language abilities cautions against over-generous interpretations of apparent *symbol processing* by man-made computation systems.

Cerebral Darwinian Processes on the Time-scale of Thought and Action

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I. Introduction

II. Shaping up thoughts is an old idea (see Calvin THE CEREBRAL SYMPHONY, 1989, ch.12)
   A. "All is but a woven web of guesses" (Xenophanes, 6th-c. B.C.)
   B. trial-and-error (Alexander Bain 1855)
   C. William James (1874, 1880) had notion of idea variants, survivals, environmental judgements
   D. Kenneth Craik (1943)
   E. and it applies to nonroutine actions of all sorts, such as creating sentences to speak, or planning for tomorrow.
   F. Generating choices, and deciding among them after shaping them up further, is absolutely central to our notions of self-determination and consciousness.

III. *Where* it happens in the brain is becoming clearer
   A. Language is perisylvian, dorsolateral frontal lobe and temporal-parietal, mostly left hemisphere.
   B. Premotor and prefrontal cortex have a lot to do with preparation for action, for both hemispheres.
   C. Narratives have a lot to do with mesial and orbital frontal cortex, both hemispheres.
   D. This narrows it down to about 1/3 of cerebral cortex...

IV. But it's a *process*, not a place. A process that might run in different places at different times.

V. Still can't say exactly *how* it happens in the brain, yet six darwinian principles are clear from species evolution and the immune response, can try applying them to cerebral processes on the milliseconds-to-minutes time scale:
   A. There is a pattern involved (typically, a string of DNA bases but it could also be a musical melody or the cerebral activity pattern associated with a thought).
   B. Copies are somehow made of this pattern (as when cells divide, but also when someone whistles a tune they've heard).
   C. Variations on the pattern sometimes occur, whether by

copying errors or shuffling the deck.

D. Competitions occur between variant patterns for occupation of a limited space (as when bluegrass and crabgrass compete for your back yard).

E. The relative success of the variant patterns is influenced by a multifaceted environment (for grass, it is hours of sunlight, soil nutrients, how often it's watered, how often it's cut, etc.).

F. And, most importantly, the process has a loop. The next generation is based on which variants survive to maturity, and that shifts the base from which the surviving variants spread new reproductive bets. This means that the variation process itself is not truly random but based on those patterns which have already survived the multifaceted environment's selection process. A spread around the currently successful is created; most variants will be worse off, but some may be even better suited.

VI. So what's the pattern? A spatiotemporal pattern of neuron activities is needed to produce an action. And spatiotemporal patterns are also thought to represent an object, the short-term holding mechanism for a memory, or an abstraction such as an idea.

A. Yet does this mean that, to recall a previous event, we have to recreate the spatiotemporal pattern of activities present at the inception, for all 10^11 neurons of cerebral cortex? Or will some subset (Hebb's cell assembly) suffice -- a cerebral code?

VII. What's the cerebral code for an apple or orange? For the genetic code, a lot of insight was gained by focussing on the copying mechanism. The double helix served to eventually identify the codon triplets for the amino acids. Similarly, my strategy has been to ask what mechanism might serve to make copies of spatiotemporal patterns in the cerebral cortex. And, of course, copying is the second essential of a darwinian process.

A. This "cerebral code" could be collapsed into a 0.5mm hexagon of cortex, though redundantly repeating in the manner of wallpaper patterns.

1. arises from mutual re-excitation among pyramidal neurons of the superficial layers of neocortex.
2. This should synchronize such pyramidal neurons in a triangular mosaic at the 0.5mm spacing of the axon terminal clusters.

3. Activity of feature detectors over several mm could, because of the screen-wrap tendencies of their triangular mosaics, tend to create an elementary spatiotemporal pattern contained in a 0.5mm hexagon of neocortex.
4. Changes in the passive connectivity of neurons within such a hexagon could allow the active spatiotemporal pattern to be recreated. And then copied elsewhere.
5. To move it from one part of the brain to another, a distant copy is made (not unlike a fax).

VIII. While some areas of cortex might be committed to full-time specialization, other areas might often support sideways copying and be erasable workspaces for darwinian shaping-up processes.

IX. As you decide between an apple and an orange, the cerebral code for apple may be having a copying competition with the one for orange, their resonance with connectivities biased by the multifaceted environment of current drives and past memories.

A. When one code has enough active copies to trip the action circuits, you may reach for the apple.

B. But the orange codes aren't entirely banished; they could linger in the background

1. as subconscious thoughts, variants on the pattern exploring for other resonances, perhaps eventually bringing tennis balls "to mind."

C. Satisfies all six essentials for a darwinian process.

D. All regions of neocortex have the essential circuitry in the superficial layers for implementing this

1. and all primate species have it (plus cats, but -- so far -- not rats)

X. The darwinian process is something of a default mechanism when there is lots of copying going on, and so we might expect a busy brain to use it

A. but also to devise shortcuts, so that faster solutions evolve for routine choices, leaving the more lengthy darwinian process to deal with

1. ambiguous pattern recognition
2. and the creation of novel output patterns (such as this sentence).

Microgenesis

Jason W. Brown, New York University Medical Center

Microgenesis is a theory of cognitive **process**, or becoming, based on inferences from patterns of pathological breakdown. The cognitive process is related to evolutionary and developmental growth trends in a number of respects, including a specification from the "archaic" to the "recent" in brain structure, and from the past to the present in an unfolding over time.

The first part of the lecture will review some principles of the theory with examples from aphasia study. The model derived from the aphasias extends to action and perception systems, and suggests that a common *bauplan* underlies mental process in different cognitive domains. Regions of the brain are constituents of "distributed planes" in a hierarchically arranged series. The "centers" of classical neurology, as revealed by lesion studies, are interpreted as moments in an actualization over this series. The unfolding proceeds from depth to surface, with each sequence constituting a minimal mind/brain state. The overlapping and iteration of states account for the continuity of mind.

The second part will explore some properties of the microgenetic process more directly. It is argued that parcellation and neoteny in morphogenesis establish, through context-item or whole-part transforms, the **pattern** and **rate** of adult cognitive process (microgenesis). Examples will be given for the evolution of lateral asymmetry and the nature of a symptom or error in adult pathology.

The third part will discuss some implications of the microgenetic account for awareness of time and duration and the nature of emergence. Subjective time depends on the cyclical and recurrent nature of the past-to-present unfolding in the mental state with duration extracted from the disparity between the decay of prior *nows* within the actual (present) state. Emergence is conceived as a specificaition of parts from wholes, with the whole always prior to the parts. The interpretation of synthetic or part-to-whole transformations is guided by the theory of (subjective) time.

Abstract

SYNERGY, SYMBIOSIS AND THE EVOLUTION OF COMPLEX SYSTEMS

Peter A. Corning, Ph.D.
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Self-organization theory has shed new light on the characteristics of biological systems and has illuminated a significant aspect of the evolutionary process. It has helped us to understand more clearly the nature of biological organization and to define some of the constraints that exist in such "anti-chaotic" systems.

What eludes the self-organization paradigm, however, is an explanation for the functional (adaptive) properties of biological systems, as well as for the remarkable evolutionary progression over the past 3.5 billion years from an amorphous mass of biochemical precursors to, ultimately, "higher" mammals.

Some proponents of the so-called Neo-Darwinian Synthesis hold that natural selection, acting upon "chance" mutations in individual genes in the context of environmental challenges, is sufficient to account for the progressive emergence of functional complexity. Other theorists maintain that some additional mechanism, or mechanisms, is required to account for the trajectory of evolution. Indeed, some theorists believe the self-directing, teleonomic properties that are evident even at the level of the genome imply a self-directed aspect to the evolutionary process itself.

In this paper, it is argued that the phenomenon of synergy -- the combined or co-operative effects which may be produced by two or more elements, nucleotides, genes, cells, parts, organs, individuals or species -- is ubiquitous in the living world and has played a major role in the evolution of functional complexity. Furthermore, this "mechanism" is compatible with, and may be able to reconcile, the various theoretical positions identified above. Synergy (a subset of which includes parasitism and symbiosis) has not somehow transcended natural selection but has instead served as an important source of creativity, of innovation at various levels of biological organization, which has been acted upon by natural selection over the course of time. The slogan "competition via co-operation" is suggestive of the relationship between synergy and the "traditional" view of natural selection.

As for the relationship between synergy and self-determination, the role of behavior as a "pacemaker" of

evolutionary change has been recognized for many years; behavioral changes often initiate changes in organism-environment relationships, which in turn set up new selection pressures. It requires only an extension of this understanding to recognize that the evolution of complexity via synergy may also have been advanced through self-determination and self-selection (positive reinforcement) by various organisms at the behavioral level.

Examples of synergy are provided to illustrate its many forms, its role at various levels of organization, and its situational and conditional properties (in conformity with the workings of both natural selection and behavioral/social selection). Some implications for the future development of self-determining systems are also discussed.

Outline

SYNERGY, SYMBIOSIS, AND SELF-ORGANIZATION IN THE EVOLUTION OF COMPLEX SYSTEMS

Peter A. Corning, Ph.D.
Institute for the Study of Complex Systems
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I. Introduction

-- Synergy in Evolution and the New Science of Complexity

II. The Evolution of Complexity as a Major Theoretical Challenge

-- Defining Complexity

-- Aristotle and the Scala Naturae

-- Genesis and Orthogenesis (Lamarck, Spencer, et al.)

-- Neo-Darwinism and its Interpreters

-- D'Arcy Thompson and Sewall Wright

-- Thermodynamics, The Second Law and Prigogine

-- Self-Organization/Autocatalysis

-- Hypercycles (Eigen)

III. Self-Organization and Complexity

-- Chaos Theory and Anti-Chaos

-- The Mathematics of Dynamical Systems: NK Models, Boolean Networks at the Edge of Chaos and Dynamic Attractors

- -- The Problem of Functional Design and Adaptation
- -- Boolean Order vs. Cybernetic Organization
- -- A Note About Gaia

IV. Symbiogenesis and Synergy in Evolution

- -- A Note About Terminology
- -- The Rediscovery of Symbiogenesis
- -- Symbiosis and Evolution
  (cf., Coevolution)
- -- The Synergism Hypothesis and its Application
  (cf., Synergetics)
- -- Synergy as a "Mechanism" of
  Evolutionary Complexification
- -- Models of Co-operation and Synergy
- -- The Mutualism-Parasitism Continuum
- -- Synergy and the Two Modes of Complexification: Integration and Differentiation
- -- Relationship to Neo-Darwinism
- -- Relationship to Self-Organization Theory
- -- Synergy as an Interdisciplinary Principle

V. Symbiosis and Synergy in Social Organization

- -- Relevant Principles
- -- Types, Classes, Categories
- -- Dynamics of Symbiosis/Synergy in Practice
- -- The Economics of Synergy

-- Synergy in the Evolution of Social Systems

-- Synergy in Human Evolution

VI. Synergy, Self-Organization and Self-Determination

-- Purposiveness and Cybernetic Self-Regulation

-- Toward a Synthesis of Functionalist/
Selectionist Mechanisms and Autogenous Mechanisms

-- Some Implications

VII. Self-Determination and the Future

-- Applying the Lessons (and Mechanisms) of Evolution

Organizational Principles in Human Behavior and Psychological Development: Analysis and Application to Motivation and Volition

Richard M. Ryan
University of Rochester

The principle of organization in biology concerns the dual tendencies toward differentiation and integration in structures and functions. Although organizational principles are widely accepted within biology (Mayr, 1982) their applicability to psychological development is still controversial. In this talk I will review the concept of organization and its meaning for psychological phenomena. Briefly I will argue that the application of biological principles to psychological processes has tended to rely heavily on evolutionary reductionism, and thus fails to underscore the most pervasive manifestations of organizational processes in *intrinsically motivated behaviors*. In other words,the innate organismic push toward differentiation and assimilation is most convincingly represented by the evident curiosity, exploratory and manipulatory tendencies, and creative playfulness of organisms within their environments. Furthermore the tendency of humans to *internalize* and *integrate* cultural mores and proscriptions is a further reflection of such organizational processes. Because both intrinsic motivation and integrated internalizations are phenomenologically characterized by the sense of autonomy or volition, I will further argue that the internal sense of volition or *self-determination* is an experiential index of the quality of psychological organization. In short, to the extent that differentiation and integration has succeeded the individual will report an *internal locus of causality* or a sense of *autonomy* with respect to their behavior. A further empirically based finding is that organizationally congruent behaviors are associated with an internal experience of *vitality*, or possession of energy. The thrust of the talk will be toward dereifying psychological organization and linking it to empirically definable motivational processes and their phenomenological accompaniments.

Organization and Self-Determination in Human Development and Behavior

Richard M. Ryan, University of Rochester

I The organization principle applied to psychological systems
- a. overview of basic principle
- b. application to biological systems
- c. application to processes of cognitive development
- d. brief review of organismic approaches to personality
- e. questions concerning the viability of organizational perspectives on behavioral regulation and personality processes

II Controversies concerning organization and self-determination in personality.
- a. conceptual difficulties in positing RinnerS regulations
- b. conceptual difficulties in positing RnaturalS functions
- c. controversy concerning the role of agency in organizational perspectives
- d. observations concerning the fragmented, and often disorganized, nature of psychological processes

III. Behavioral processes that reflect organizational tendencies
- a. the RdiscoveryS of intrinsically motivated behavior (IMB)
  - . role of IMB in psychological development
  - . research on the conditions supporting IMBs
  - . in what way are IMBs invariantly Rself-determinedS
- b. self-determination in non-intrinsically motivated action
  - . phenomenology and attributional processes associated with self-determination
  - . the concept of internalization and its relation to organization (integration) and self-determination
  - . some studies on the continuum underlying internalization processes
  - . attachment processes and internalization
  - . the role of internalization in the transmission and organization of culture

V. Overview of the connection between phenomenological and structural descriptions of integration and self-determination.
- a. PolanyiUs active center
- b. the perceived locus of causality for action and its relation to organization and vitality

c. specific factors affecting self-determination processes

VI. Motivational theories and the sociobiological approach
a. what sociobiology has gleaned from evolutionary theories
b. the mistaken priority of drives as organizers of behavior
c. psychological needs associated with organization
d. implications of an organizational perspective on evolution and behavior
e. implications of organizational perspectives on behavior for sociology

VII. Summary, conclusions, and prelude to Deci talk

Effects of the Social Contexts on Self-Determination

Edward L. Deci
University of Rochester

The organization principle, inherent in the nature of life, is a fundamental property of *self*, which functions most effectively under conditions that support satisfaction of the intrinsic psychological needs for competence, autonomy, and relatedness. To remain healthy, individuals must feel: (1) efficacious with respect to their surrounds, (2) agentic in their actions, and (3) both loved and loving. Although the fact of these needs could be understood in evolutionary terms, I will address the issue on empirical grounds by reviewing evidence that when people have the opportunity to satisfy these three psychological needs, they develop greater coherence of self, evidence enhanced self-determination, and function more effectively. Conversely, when they are denied such opportunities, they become less effective and display a variety of symptoms.

The postulate that these needs exist and are fundamental is supported by the fact that they have utility for integrating a variety of experiments that detail how the social context affects motivation, development, and performance. Specifically, contexts (1) that provide optimal challenge and relevant feedback (thus supporting competence), (2) that provide choice, acknowledge one's perspective, and encourage self-initiation (thus supporting autonomy), and (3) that provide and respond to warmth and attention (thus supporting relatedness) have been found to increase intrinsic motivation, to facilitate internalization (i.e., organismic integration), to promote creativity and effective problem solving, and to enhance both psychological and physical health. In contrast, contexts that deny any of these necessary nutriments for the development of self have been found detrimental to these indicators of effective functioning.

Effects of Social Context on Self-Determination

Edward L. Deci, University of Rochester

I. The Organization Principle in Psychic Life

- My comments follow directly those by Richard M. Ryan.
- The organization principle is integrally related to the concepts of agency and self.
- Self represents a subset of all psychological processes and structures. It results from the ongoing integration of values, attitudes, beliefs, and regulatory processes with one's intrinsic or core self.
- Self is thus the ongoing developmental outcome of the organization principle.
- Self is, however, also an input to organization for as one acts agentically, from the self (rather than from fractionated aspects of the psyche), organization is facilitated.

II. The Motivational Basis of Organization and Self

- We have studied organization and self-determination at the psychological level using the standard empirical methods of psychology.
- We begin with a descriptive continuum that characterizes behavior in terms of the degree to which it is controlled versus self-determined. Control refers to feeling pressured or coerced to act, whereas self-determination refers to feeling volitional and choiceful while acting.
- Self-determined actions are regulated by the integrated self,
whereas controlled actions are coerced by nonintegrated forces.
- Behaviors that are self-determined are motivated either by (1)
intrinsic motivation or (2) regulatory processes that were initially external but have been integrated with the self.
- Intrinsic motivation underlies the natural, spontaneous, self-
determined activity, readily apparent in young children. It operates in the service of the three innate psychological needs for (1) competence, (2) relatedness, and (3) autonomy.
- People internalize and integrate the regulation of socially sanctioned activities that are not spontaneous in order to

(1) be competent in the social world, (2) feel related to others, and (3) feel autonomous and volitional in those activities that allow social efficacy.

III. Social Contextual Influences on Organization and Self-Determination.

- Although intrinsically motivated activity and organismic integration are both natural processes, they require contextual nutriments.
- The necessary nutriments are the ones that allow people to feel (1) competent, (2) related to others, and (3) autonomous in their actions.
- Social contexts will facilitate organization and self-determination to the extent that they support the satisfaction of all three psychological needs. They will impair organization and self-determination to the extent that they thwart satisfaction of one or more of these needs.
- Because self-determination is associated with greater creativity, deeper processing of information, enhanced cognitive flexibility, and a more positive emotional tone, the effects of the environment on these human qualities are understood to be mediated by organization and self-determination (and thus by the satisfaction of the three basic psychological needs).
- Aspects of the social environment affect both (1) the degree to which an immediate behavior is self-determined and (2) the degree to which the ongoing developmental process of organismic integration will occur. Because organismic integration promotes integration of the self, the social context ongoingly influences the development of self-determination as an individual difference in personality.
- Research has shown that interpersonal contexts that (1) provide optimal challenges and relevant, noncritical feedback (thus supporting competence), (2) provide and respond to interpersonal warmth and attention (thus supporting relatedness), and (3) provide choice, take an internal frame of reference, and encourage self-initiation (thus supporting autonomy) enhance intrinsic motivation, promote integration, and facilitate effective self-determined functioning.

Towards the evolution of communication

David H. Ackley
Bellcore

Michael L. Littman
Bellcore & Brown University

Models of Darwinian evolution typically emphasize individual fitness, whereas models of communication to accomplish a task tend to focus on the communicating parties as a group. To model the evolution of communication, one must somehow straddle these two levels of description and deal with the contentious issue of 'group selection'. Computer simulations have been used to illustrate the evolution of communication, but have so far always employed 'fitness functions' that rewarded individual speech acts based on the consequent behavior of the listener. Although interesting in their own right, such models beg the question of individual versus group selection by, in effect, combining speaker and listener into a single 'virtual individual' for purposes of fitness evaluation. In this work we asked: Can useful communication evolve if individuals can 'speak' and 'hear', but their fitness depends only on what they *do*, and not what they *say*?

We divided a large simulated population into small, semi-isolated groups, and implemented a very simple environmental model that provided opportunities for useful communication within each subpopulation. We expected that this division of the population would lead to close genetic kinship among the individuals of each subpopulation, and hoped that 'genetic altruism' would then be enough to favor the evolution of communication.

In case studies, we observed that subpopulations capable of effective communication did evolve, but, unexpectedly, we also discovered that such subpopulations were often unstable. We found that when individual fitness is the only criteria for reproductive success, selection pressure preferentially rewards 'parasites' that 'understand' (i.e., 'react in an individually fit way to') what is said by others, but do not themselves 'speak truthfully' (i.e., 'employ the stimulus-to-speech conventions used by the rest of the subpopulation'). Such a cheater does not display altruism even though it usually shares the vast majority of its genes with the rest of the subpopulation, and as the parasite reproduces, effective communication within the subpopulation dwindles, and the fitnesses of all the individuals in the subpopulation drop dramatically.

Evolution in Other Universes

by Tom Ray

Our concepts of biology, evolution and complexity are constrained by having observed only a single instance of life, life on Earth. A truly comparative biology is needed to extend these concepts. Because we can not observe life on other planets, we are left with the alternative of creating artificial life forms on Earth. I will discuss the approach of inoculating evolution by natural selection into the medium of the digital computer. This is not a physical/chemical medium, it is a logical/informational medium. Thus these new instances of evolution are not subject to the same physical laws as organic evolution (e.g., the laws of thermodynamics), and therefore exist in what amounts to another universe, governed by the ``physical laws" of the logic of the computer. This exercise gives us a broader perspective on what evolution is and what it does.

This evolutionary approach to synthetic biology consists of inoculating the process of evolution by natural selection into an artificial medium. Evolution is then allowed to find the natural forms of living organisms in the artificial medium. These are not models of life, but independent instances of life. In this approach we strive to understand and respect the natural form of the artificial medium, to facilitate the process of evolution in generating forms that are adapted to the medium, and to let evolution find forms and processes that naturally exploit the possibilities inherent in the medium. Examples will be cited of synthetic biology embedded in the computational medium, where in addition to being an exercise in experimental comparative evolutionary biology, it is also a possible means of harnessing the evolutionary process for the production of complex computer software.

Initial experiments show that evolution leads quickly to the emergence of an ecosystem of digital organisms which evolve interactions of exploitation, defense, cooperation, cheating, etc. Evolution generates a long series of these interactions. In addition, evolution generates tremendous optimizations, sometimes achieved through great reductions in the size of the code, at other times through great increases in its complexity.

Comparisons of four different digital ``universes" shows striking differences in the mode and degree of evolution, depending on the underlying ``physics". Two universes show gradualism, one punctuated equilibrium, and one punctuated gradualism. Those exhibiting punctuations achieve greater degrees of evolution.

Further analysis showed that the highest scoring of these metastable communicating subpopulations are 'obligate social' in the sense that when they are tested in an environment where communication is blocked, they score very poorly, much worse than subpopulations that had not evolved a dependence upon communication. The conflict between the fitness gains possible through communication and the consequent vulnerability to parasitism appear to favor the emergence of 'facultative social' individuals that balance the two influences: They employ communication to advantage when among their own kind in a communication-supporting world --- though not as effectively as the obligate socials --- but even when communication is blocked, these 'cautious communicators' manage to achieve the maximum fitness possible for non-communicators.

In this workshop presentation, I will outline prior work in computer models of the evolution of communication, present the model we've studied most deeply, discuss the simulation results, and show some video that depicts the short- and long-term dynamics of the system.

[draft manuscript for workshop on self-determination in developing and evolving systems, Boston, MA, January 6-9, 1994; version of 12/1/93. Comments are welcome!]

Level-invariant processes of self-determination: Scenario, examples, and implications

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Abstract

A scenario for evolution and development summarizes ways in which a living system finds and keeps good goals. The system consists of material units that catalyze a network of processes. The connectivity of this network evolves to match the pattern of correlations among constraints on the system. This scenario is evident in the evolution of metabolism, and in morphological and psychological development. Selection for flexibility favors the evolution of matching, through dynamic modules that deal with invariant aspects of the system's world. Selection for reliability stabilizes modules by favoring more parallel, fewer serial, and more reliable component processes. A capacity for regression and regeneration allows a system to operate reliably, but also to respond to new constraints.

Introduction

What are the goals of a living system? How does it find and keep them? A living system is a multifunctional catalyst: It can facilitate the occurrence of a large set of processes, at many levels of organization. A biochemical reaction is a low-level process; the division of a cell and the thinking of a thought are processes at higher levels. Each process transforms a set of inputs to a set of outputs, which is its goal. The laws of nature constrain the available processes and their goals. In this sense a system can find goals but not create them. An organism can find a niche that it might match; it does not create the niche.

To ask how a system finds its goals is to ask how it comes to perform processes that generate particular outputs, given particular inputs. Good goals for the system give it high benefit at low risk; bad goals give low benefit at high risk. Impossible goals do not correspond to an attainable final state. (Possible and impossible, good and bad, are obviously extremes in a spectrum of possibilities, benefits, and risks.) Because the resources of the system are limited, it must find good goals and avoid impossible or bad ones. We want to understand how a system finds good goals, and how to characterize the organization of such a system.

A scenario can describe the generation of goals at various levels of organization and various time scales. In this scenario a living system consists of units that can perform low-level processes. The units may be enzymes, genes, cells, or groups of cells. The units catalyze processes, and the outputs activate and inhibit other processes. These unit-level processes and interactions form a network of processes. During evolution the connectivity of the network changes, changing the set of higher- level processes that can be performed – and the goals of the system.

We'll use this scenario to ask, What are the characteristics of a network that typically finds good goals? Why do most changes in its connectivity leave it with good goals? As we'll see, the connectivity of networks with good goals seems to obey a principle of matching: Unit-level processes are coupled to achieve goals that correspond to invariant aspects of the system's world. Dissociable clusters of coupled processes deal with dissociable invariants. For example, the colors and forms of objects are dissociable aspects of the visual world. Correspondingly, the neural pathways that process them are at least partially dissociable: A color-blind person can see forms, and a very myopic person can see colors but not forms (Clarke and Mittenthal, 1992; Mittenthal et al., 1992).

A process with inputs and outputs dissociable from other aspects of the world will be called a module. Note that this is a dynamic module, a process with a characteristic dynamic, as distinguished from a module that is a material structure, such as the units in the preceding scenario. I think a dynamic module corresponds to an object in object-oriented programming, but I don't know much about the latter.

By looking at the evolution of metabolism, we'll see that a system with matching and modules can find and keep good goals. In metabolism and in development, selection for flexible and reliable catalysis favors the evolution of modules.

The evolution of metabolism

Let's see how intermediary metabolism may have evolved. Metabolism maintains the pools of key metabolites that a cell needs for its activities – monomers for polymerization, energy carriers, reducing agents, and cofactors for enzymes. ¡figure from Rawn¿ Metabolism synthesizes these small molecules from each other and from molecules outside the cell, through a network of enzyme-mediated reactions.

Early in evolution the sequence of amino acids in enzymes became encoded in DNA, the enzymes became encapsulated in a membrane, and cells became capable of self-reproduction. We will not be concerned with these processes, other than to recognize that they created a population of metabolic networks. It has been suggested that early enzymes were short peptides, which catalyze reactions at low speed and with low specificity. Several enzymes of low specificity may have catalyzed each reaction. This overlapping redundancy makes the performance of a network of reactions relatively reliable against deletion of an enzyme. Over time, variant enzymes appeared, and competition among the networks that used them changed the types of networks present.

The networks that survive in the competition must have met performance criteria of flexibility, reliability, economy, and speed. In a variable environment a good network is flexible; it can adjust the concentrations of different key metabolites independently, in a way responsive to the demand for them and to the supply of substrates. Selection for speed favored enzymes that were longer peptides with greater specificity, which can catalyze reactions faster. However, greater specificity spoils the reliability achieved in the early networks with overlapping redundancy. Reliability could be sus-

tained if there were more types of longer peptides. Processes of mutation, including duplication and divergence of genes and exon shuffling, produced this diversity. However, the number and length of peptides that a cell can make is constrained, by errors that limit the coding capacity of the DNA and by the solubility of the peptides. These constraints impose a selection pressure for economy.

My collaborators and I explored whether a network that performs with flexibility, reliability, speed, and economy is likely to have a characteristic structure (Mittenthal et al., 1993). We found that this is so in an artificially simplified model: A favorable network is likely to have a hub-and-branch structure, in which key metabolites are degraded to a hub of interconvertible metabolites, and can be synthesized from the hub. Economy in the number of enzymes is improved if pathways between key metabolites and the hub share intermediate metabolites – that is, if the pathways branch. We are now investigating whether an optimal structure for more realistic networks is also a hub-and-branch structure; we expect so.

A hub-and-branch structure obeys the principle of matching: The coupling among processes within the system matches the correlation among its goals. The goal of providing diverse carbon skeletons is dissociable from the goal of decorating these skeletons with particular side groups in the key metabolites. The separation of hub from branches corresponds to this dissociation; the hub makes carbon skeletons and provides energy and reducing power, while the branches synthesize and degrade the key metabolites. In a branching pathway each branch operates as a module: Its activity, consuming its substrate and making its product, can be adjusted independently of the activity of other modules, by changes in the concentrations of its product and substrate. The entire branching pathway is a higher-level module; it corresponds to the existence of families of key metabolites, which can all be synthesized from the same starting compound using relatively few enzymes. The entire network is a module at a still higher level; its task is to maintain stable pools of key metabolites.

The evolution of metabolism illustrates our scenario. A metabolism has a set of units – enzymes – which catalyze unit- level processes – biochemical reactions. As evolution occurs, variant units appear – peptides with different lengths, amino acid sequences, affinities for binding ligands, and catalytic specificities. Sets of peptides with catalytic activities that match the pattern of functional demands survive selection. The outcome is a network with a characteristic connectivity – for metabolism, a hub and branch structure. This structure can operate as a hierarchy of modules.

Selection for flexibility favors modules and matching in development.

Metabolism shows us that a network with modules and matching can perform well and have good goals. Does a change in the connectivity of such a network usually leave modules and matching? It's easier to ask this question at a higher level than metabolism, where more diverse networks have evolved. So, we shall look at the networks that mediate the development of a multicellular organism from a fertilized egg. We shall see that modules and matching can persist through changes in connectivity. Some changes leave modules intact but displace them in space or time, modify their output, change their reliability, or couple them into new, higher-level modules. Mutations naturally tend to destroy extant modules, but can couple lower-level processes in new combinations. Selection can favor the retention of old modules, or of new modules formed by novel associations of processes.

A metabolism must interconvert key metabolites with flexibility; this requirement implies the dissociable modules that are evident in a hub-and-branch metabolic network. Similarly, developmental modules can give evolutionary flexibility. In an embryo all the organs needed for postembryonic life must develop. In different but related species, homologous organs differ – in size relative to other organs, in mass, in shape, in biochemical specialization. Thus the organs at hatching represent dissociable goals for development, to some extent. (Clearly some aspects are dissociable and others are not. The specific processes that generate a pancreas are probably unrelated to those that generate an arm, except to the slight extent that they share or compete for the resources available to the embryo. However, the developing bones and muscles in the arm must interact, so that the muscles attach firmly to the bones, and the bones can support the forces that the muscles exert.

Because aspects of organs represent dissociable goals, the principle of matching suggests that a module generates a dissociable aspect of an organ, and this seems to be the case. Fertilization is a module at the cellular level. Embryonic induction activates a module at the tissue level; this module, called a morphogenetic field, generates an organ. The dissociability of modules within a morphogenetic field is evident in experiments that make hybrid branching organs, such as a salivary gland that secretes milk (); here the dissociation is between processes that generate tissue-level morphology and processes that transport molecules across the epithelium lining the organ. The dissociability of modules that make different organs is evident in

evolutionary changes in the timing and site of development of organs, and in their structure and activity. [figures illustrating heterochrony, homeiosis, heterotopy, ...]

The psychological development of a human also proceeds through modules and matching. In a developing organ cells proliferate, interact and differentiate. Similarly, in a baby thoughts and feelings proliferate and diversify, through their interaction with each other and with new experience. As the baby experiences different aspects of an invariant phenomenon, the corresponding thoughts and feelings become coupled in a module. This module is a dynamic representation of the invariant, mediated by assemblies of neurons, endocrine cells, and other cells. For example, a baby experiences the volition to move his arm, and then receives proprioceptive, tactile, and visual feedback from the movement. If his mother moves her arm he may see the movement, but he will not experience volition or mechanosensory feedback (Stern, 1990). Thus dissociable experiences will contribute to his developing distinct representations of himself and his mother. In this way a representation of his world develops, in which associations among thoughts and feelings match invariants, and dissociable phenomena correspond to distinct associations.

The molecular basis of modules, matching, and their flexibility in development

How does a developmental module work? It is a cascade of processes mediated by a set of macromolecules, each of which has inputs and outputs. The first macromolecules to be activated are receptors which bind a small set of messenger molecules – neurotransmitters, hormones, molecules of the extracellular matrix, molecules in the membranes of neighboring cells. These receptors catalyze the formation of other messengers, which activate or inhibit other receptors. A cascade of receptor- messenger interactions follows. Eventually messengers alter the activity of genes which encode the synthesis of proteins that may be messengers, but that have other activities. ¡figure illustrating network that implements a module: SciAm 9/93 on response of a mast cell in allergy.¿ These proteins can cooperate to change a cell's secretions, its rate of mitosis, its motility, and its adhesive affinity for other cells. Some outputs of the module can be used as inputs to other modules. Thus the entire network of processes that generate an embryo is a branching structure that includes many modules, with some anastomoses

between them, radiating from the process of fertilization. It is a kind of hub-and-branch network. []

During evolution developmental networks change; modules are formed, lost, or modified. These changes result from changes in the set of messengers, receptors, and other proteins that cells can make. Duplication and divergence, exon shuffling, and other mutations produce families of proteins. The processes of mutation are among the self-organizing activities through which organisms offer variant forms for selection. Changes in proteins allow changes in the coupling among unit-level processes, which in turn alter the available set of higher-level modules.

Developmental modules probably evolved in ways analogous to the evolution of modules in metabolism. When a new pattern of coupling among processes appears, it may catalyze a new higher- level process in a relatively weak and nonspecific way, and not in a particularly favorable context (location in the body, time of occurrence, set of activating inputs). Nevertheless, the capacity to perform a new process may provide the organism with a significant selective advantage. Gradually variations improve the performance of the module. It can become more responsive to indicators of the need for its action, and so can be used in a more appropriate context. For example, mesoderm cells will form blood vessels with better functionality if they do so in response to a lack of oxygen.

Selection for reliability stabilizes modular connectivity

If the connectivity of a network can change, why do modules function reliably? To address this question it is useful to consider first changes in the access to modules, and then changes within modules. The access to a module occurs via the messengers that activate or inhibit its use by binding to receptors. The effects of different combinations of messengers on a module specify its access code. A few kinds of messengers suffice to activate a module, so the conditions for activating it in a new context are easily met. For example, in the genetic code a triplet codon of bases specifies the amino acid to be added to a growing polypeptide chain; each triplet is used in many contexts. A low-level module tends to be localized in space and time; it can be displaced and still remain under control by a higher-level module which is less localized. (The same codon can be used at various positions along a peptide.)

Changes in the access code of a module tend to leave the system functional because a module is often a member of a class of modules, in which a set of inputs elicits a set of outputs. Within this class a similarity code often operates, so that similar inputs elicit similar outputs. The triplet code is a similarity code. Similar codons can specify the same amino acid; this exemplifies an equivalence class of inputs, all of which generate the same output. If one of the three bases in a codon is changed, typically the new codon is in the same equivalence class as the old, or both encode chemically similar amino acids (e.g. both hydrophobic). These features of modules – few inputs, invariance to spatial or temporal displacement, and similarity coding with equivalence classes – increase the reliability with which inputs of a class elicit outputs adequate to meet goals.

To ask whether the processes within a module tend to be stable against mutation, let us look at a simplified model. Consider a module that is a network of gene activities, one of an ensemble of networks that can be interconverted by mutation (Clarke and Mittenthal, 1992; Clarke et al., 1994). Each network can synthesize four proteins, A, B, C, and D, which associate through the dimers AB and CD to form the tetramer ABCD. Each gene has a cis-regulatory element that can bind one transcription factor, and a coding region that encodes the synthesis of a protein, a transcription factor or a monomer. When the gene is activated, the protein is synthesized. There are no inhibitory transcription factors, and messenger RNA is neglected.

This model is an example of our scenario; the units are genes, each of which generates an output in response to an input. The genes form a network because a transcription factor encoded by one gene can activate another gene. Genes are activated at four sequential levels. Transcription factors of a different family are synthesized at each level, and bind only to genes at the following level. [] In different networks of the ensemble, corresponding genes encode the same protein, but they may bind different transcription factors of a family. Each of 6 genes binds one of two transcription factors, so there are 64 possible networks in the ensemble. These fall into 8 equivalence classes. The networks in an equivalence class have equivalent connectivity but use different transcription factors. Each equivalence class represents a macroscopic pattern of connectivity.

Four of the equivalence classes obey the principle of matching: A module makes each of the two dimers, in that coupling of genes through one transcription factor coordinates the making of the monomers that associate in the dimer. ¡¿ This coupling offers a selective advantage because the genes do not respond reliably to their inputs, and the lifetimes of the monomers

are finite, so networks without matching will produce tetramers successfully at a lower rate than those with matching. The reliability of a network is the probability that it generates a tetramer in response to activation; the reliability is a measure of the fitness of the network, the selective advantage it provides. Among the remaining four equivalence classes, in two a module makes only one of the two dimers. The remaining two classes are nonmodular, in that the coupling among genes does not match the association among monomers. []

Of the 64 networks, 1/4 make both dimers with modules, 1/2 make only one dimer with a module, and 1/4 do not have modules. If making at least one dimer with a module offers a selective advantage, the majority of mutations will leave a network with some selective advantage over a nonmodular network. However, selection – a difference of fitness among networks – is required to sustain matching. Starting from arbitrary initial conditions, with a constant environment that favors synthesis of the tetramer at a high rate, a population of networks evolves to an asymptotic distribution of networks over the equivalence classes. [] At a realistically low mutation rate a small difference in fitness will produce an asymptotic distribution in which nearly all of the networks make both dimers with modules. [] Thus, despite mutation, a modular organization that matches goals can be sustained.

Selection for reliability of performance: More parallel, fewer serial, and more reliable component processes.

An extension of the preceding model helps us to think about changes within a module that will tend to increase its reliability. As before, we assume that each gene is not wholly reliable in responding to the presence of activating messengers by producing its protein. Suppose there are more levels of genes that make transcription factors, and more genes per level. In each gene the cis-regulatory region can bind several transcription factors; some of these can activate the gene, while others may inhibit it. These changes make the network more realistic.

Activation of the module elicits production of the tetramer. This process is analogous to the conduction of electric current in a wire, by the passage of an electron along it. The reliability of the module is analogous to the conductance of the wire, and depends on factors analogous to the cross-sectional area, length, and resistivity of the wire. Increasing the cross-

sectional area is analogous to redundancy in the module – to increasing the number of paths through which the initial activation can elicit a tetramer. Decreasing the length of the wire corresponds to decreasing the number of processes in sequence in the module, and to shortening the spatial interval within which these processes occur. Decreasing the resistivity of the wire corresponds to increasing the reliability of component processes in the module – for example, by error correction. (Note that adding an error-correcting process in series after an unreliable process increases its reliability. This is an exception to the general case, that the reliability of sequential processes is lower, the more processes there are in series.) [examples]

Selection for reliability with flexibility: Regeneration

The preceding model does not show how likely it is that new, different modules will arise by mutation. It seems likely that higher-level modules will evolve readily if selection has favored reliable lower-level modules, which can be coupled in many ways. But how can such a system deal with a constraint that is not compatible with its lower-level modules? One strategy is evident: If the normal operation of the system produces an inadequate output, the system can regress partially to an earlier stage of evolution or development, and then can generate a modified output. This strategy is used by bacteria which must grow on a substrate that they can not metabolize. If a lac- strain must grow on lactose, the bacteria may relax error correction, so that genes and enzymes containing many errors are produced. Some enzymes may have broader specificity (and lower activity) than the normal ones, and so may offer a pathway to metabolize lactose. If this occurs, selection can again tune the system toward higher specificity and activity, as occurred during its initial evolution.

Some developmental modules use this strategy of regression and redifferentiation if their operation is perturbed. For example, consider a simplified caricature of the process that generates a limb. We treat the developing limb bud as an elongating column of cells, several cells wide; cells are the units in this scenario. [] Each cell has a state, labelled with a letter. All cells in a cross-section of the limb have the same state, but cells differ along the length of the limb. During development a cell undergoes transitions of state that depend on its current state and the states of its nearby neighbors, as the cells activate and inhibit each other's proliferation and differentiation.

As cells divide, the new cells participate in the state transitions. The set of rules for state transitions, together with initial and boundary conditions for the cells subject to these rules, constitute a morphogenetic field. (This model has been generalized to a limb formed from a cell sheet or mass in which the states of cells vary within a cross-section. It has also been used to interpret the regeneration of multiple limbs after diverse traumas; see Bryant, Bryant and Muneoka, 1986; Winfree; Mittenthal, 1981; Stocum.)

Simple transition rules might look like this: Suppose the cells at the free (distal) end of the column always take the boundary state I. (The cell at the proximal end of the line, with state A, remains connected to other cells not considered here.) Each cell takes a state intermediate between the states of its neighbors. New cells stop being generated when each unit has neighbors with states that are adjacent in the alphabet. With these transition rules, a limb with the sequence of states ABCDEFGHI normally develops. A wound disrupts the normal patterns of activation and inhibition among cells at the wound margin, so that their states are destabilized. Consequently they dedifferentiate (regress) and then redifferentiate with states appropriate to their neighbors. If the wound is small, this process can restore a normal pattern of cell states.

However, regression and redifferentiation do not always produce a normal output. If a wound is large and heals slowly, cells at its margin may redifferentiate with the boundary state I, because they seem to be at a new distal boundary of the limb. With this new boundary condition the self-generative processes can make an abnormal limb that terminates in multiple tips (cf. Bohn, 1965; Shelton et al., 1981). [] Here the module catalyzes a generative process with abnormal boundary conditions to produce a limb that is locally normal but globally abnormal. Regression and redifferentiation also occur in psychological development, after a child experiences trauma repeatedly. The abused child tends to revert to more immature behaviors, with decreases in imaginative activity, play, exploratory behavior, and social activity. This regression may be a necessary prerequisite to subsequent development, even if a dysfunctional self-representation develops. The preceding model for the morphogenesis of multiple structures has suggested a model for the development of multiple personalities (Woollcott and Mittenthal, in preparation).

In these developmental examples of regeneration the modules themselves do not change, unlike the example of bacterial re- evolution. However, the wound produced by physical or psychological trauma is a functionally inadequate consequence of development. Regression and regeneration may

produce a more adequate outcome.

Discussion

We have inquired how a living system comes to perform processes that favor its persistence. The preceding examples suggest that at diverse levels of organization, a network of processes can operate with flexibility and reliability if it has modules and matching. Its reliability can increase by increasing parallel processing, decreasing the number of processes in series, or increasing the reliability of processes. Regression of the system to an earlier state allows it to function reliably with a given set of modules, but to restructure modules in adverse conditions.

More generally, why is it useful to recognize the occurrence of modules and matching? A theory about the organization of living systems must characterize their form and content. The principle of matching characterizes the form of a biological system at an abstract level, the coupling among processes. The match between coupling and constraints seems to be independent of the level of organization and the time scale. By contrast, the content of a system is specific to the state and dynamics of its material units, and varies with level of organization and with time. Thus we can distinguish the invariant aspects of living systems from their particulars, and so see unifying themes pervading the diversity of life.

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Single Cell Agents to Multi-Cell Hierarchies: Constraints, Mechanisms and Simulations

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Abstract: This study attempts to gain insights into a process that allows single cell agents to evolve to multi-cell hierarchies. I have tried to apply minimal, real world assumptions and constraints to a simulation of this process. Assumptions are made about conservation principles, entropy, cost-benefit mechanisms for cell agents, agent-to-agent transactions, and mechanisms of reproduction. The simulations use constraints on the generation, distribution and consumption of resources as well as regulatory mechanisms. The system demonstrates a dynamic balance between the self-interest of the individual and the group as well as between self-directed behavior and natural selection pressures. A key feature of the system is how catalytic or synergistic interactions in hierarchies make them more efficient and stable. The simulation results may generate insights relevant at all levels of life from cell biology to human culture.

Introduction

Much of life is involved with relationships and transactions, whether it is evolving multi-cell organisms, growing a multi-tissue embryo, surviving within ecosystems or developing cultural institutions. How and why do multicell organisms evolve? Many Artificial Life models attempt to mimic gene recombination, gene mutation and natural selection phenomena to evolve agents which optimize some environmental fitness function (Forest, 1991) These models have had great success in both classification and optimization problems. For these problems, there has been no need to evolve agents that are more complex than the initial agents that began the simulation and when they reach an asymptotic level of functionality, their evolution hovers around the asymptotic level. So why are multi-cellular agents created and what benefit do they have over single cell agents? To study these phenomena, I built a simulation environment with constraints and assumptions that could be taken from any level of life. To be effective in cutting across different levels of life, I attempted to create a design that transcends the requirements of any particular level of life. The typical Darwinian model implicitly treats agents as relatively passive, in the sense that the agent is made to mutate and recombine without choice and at random (Wesson, 1991).

What choices are available to agents with no nervous system and what are the effects of such choices? I make a number of assumptions that are fundamentally different than most Artificial Life models. In my design, an agent's evolution has an important self-determined component. How this relates to choice will become evident later in this paper.

My design assumptions begin with some conservation principles and entropy. Further assumptions rely on the defining characteristics of relationships and transactions between agents. Finally, I will rely strongly on an endogenous self-interest function.

Design Constraints and Assumptions

The overall fitness selection function will be based on the conservation of energy. I assume that various quantities of energy are available in the environment at various times. I further assume that an agent needs to have energy to live. To be used by an agent, energy must be harvested from the environment or from transactions with other agents. The harvesting is done with less than ideal efficiency. This fitness function does not mean life or death at every reproduction. Rather, the agent leads an eventful life that will impact its ability to sustain and reproduce itself. As I shall show, what an agent does during its lifetime can affect its evolution as much as what it is born with.

All agents have a finite life time, based on the assumption that internal support functions wear down. All agents have the ability to reproduce by cloning and splitting. When an agent gets enough energy to reach a threshold, it will split into two equal agents with half the energy and the two children have the same abilities and identification as the parent. So far, there is no mechanism for innovation. With only these assumptions to run the system, agents in this system will either die off or get to some stable number or reproduce to saturate the space.

What causes agents to change or innovate? Entropy will mainly create mistakes in the support functions of the agent. The probability of a mistake turning out to be better than the original is practically non-existent. It would be like adding and removing marks in a blueprint to make a better house. Random mutation as a mechanism for innovation has some implicit assumptions that need to be better understood. First, most random changes of the genotype will result in non-viable agents. For a mutation in a genotype to create a viable agent, the mutation should either not adversely affect the dependencies of the gene or affect a relatively independent gene module. With random mutation, there may be some small chance of making a better organism. But better in what sense? It could be more efficient in harvesting energy or live longer or reproduce faster. What is the pressure to change when an organism is surviving and reproducing successfully?

I propose that the alternative to a Darwinian model is an endogenous self-interest function. In its simplest form, "if something is good, more is better". For the current design, the self-interest function is getting more energy. In a real world, there are many contingencies that can affect an agent's ability to get energy. An agent can improve its self-interest by minimal planning of actions based on consequences. Planning may seem too complex for simple agents, but I will show a primitive form of planning that requires minimal structure.

An agent can also improve its self-interest by cost-beneficial transactions with other agents. Cost-beneficial transactions are more likely with agents that have complementary abilities and needs, since the chance that both agents in a transaction can perceive self-interest, is increased. With self-interest functions and cost-beneficial transactions, groups emerge when the individuals in the group all perceive it is to their benefit to be in a group. What benefits can a group provide that individuals need and at what cost? The answer provided here will turn out to be self-interest efficiency and stability from synergistic transactions.

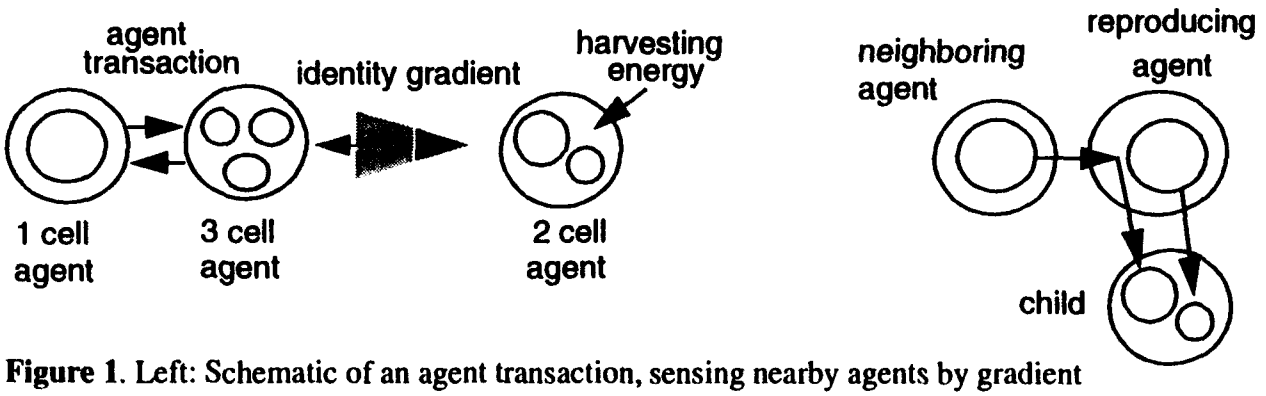

Figure 1. Left: Schematic of an agent transaction, sensing nearby agents by gradient and harvesting energy. Right: Reproduction of multi-cell child from touching agents by conjugation

Design

The design of the simulation consists of a two-dimensional matrix of unit places with agents that move, reproduce, harvest and transact with other agents. The self-interest function is to get as much energy as possible. Energy is supplied to the empty places in the matrix by an environmental "sun". Agents can not create their own energy. Instead, they gain energy by harvesting it and through agent transactions. Agents use up energy in their behavior. One allowable transaction with other agents consist of taking energy from friendly agents that have more energy. When two friendly agents touch they will split the difference of their energies. Other types of transactions include attacking hostile agents to take all their energy and defending against hostile agents trying to take too much energy. Agents expend energy by just existing, moving, defending and trying to attack without success. If an agent is harvesting on background energy, it will not move, during the harvest.

Agents have primitive sensory and response abilities. They can sense the identity of other agents, both when they touch and when they are nearby. I assume that sensations of nearby agents occur through diffusion gradients of identifying material. A beneficial transaction will cause the agent to stay in place, while a costly transaction will cause the agent to move away. But, there are some more response abilities that have a greater effect on behavior. Being able to sense agents nearby, opens the opportunity to anticipate good or bad consequences and act on them before they take place. Anticipation can enhance the efficiency of self-interest. I allow agents to have the ability to anticipate. It comes from a primitive type of memory and learning. The basic requirements of memory is the ability to make a response even after a stimulus is turned off.

I assume that agents also have a primitive learning ability in which beneficial transactions cause an agent to move towards and costly transactions cause an agent to move away from the agent originating the transaction and any other agents nearby. One important result of this learning is that agents will move toward nearby agents that did not directly affect the consequence of a transaction. It assumes that on average, the collection of nearby agents represent either a benefit or cost as a whole. Another way of looking at, is a sort of primitive transitivity effect. If an agent touches "A" and "A" is good and "B" is close to "A", then "B" must be good. In the future, if the agent senses only "B" nearby, it will move towards "B". This can have either good or bad

consequences. If "B" is actually good, then the agent has learned to generalize cause and effect in its environment. If "B" is bad, then the agent has made a mistake. Obviously, this learning requires forgetting to prevent perseverating on mistakes. The design of agent transactions is summarized in Figure 1, left.

The assumptions I have made thus far will cause some type of group forming behavior and the group will be of benefit to all the members that formed it. What makes the group form is that agents which have had beneficial transactions with friends, seek to be near these friends, if the friends can be sensed nearby. A group can split if either competition for friendship is created by two nearby friends or some enemy is sensed nearby. What holds the group together is that each member is benefiting from the pair-wise transactions in the group. But how can the children of these agents, benefit from this grouping effect? If the agents simply get cloned and split into two children, they would have to relearn what their parents learned to reform a group similar to their parents. Is there a more efficient way to inherit the benefit of the group? I suggest a way.

When a member of a group has enough energy to reach a threshold and reproduce, it is allowed to reproduce by conjugation. The reproducing agent internalizes copies of the genes of its contacting neighbors and then splits into a child, as shown in Figure 1, right. Thus the children have the blueprint for the functionality of part or all of a successful group. I assume the abilities of this multicell agent are similar to the abilities of a collection of single cell agents with the same genes. Thus, I suggest that the benefit of passing on complex organisms in evolution is to build on the behavioral success of a group of organisms, one level simpler. Success here is defined as satisfying an agent's need for a stable supply of energy. This scenario creates a feedback loop: Agents make behavioral choices based on real environmental contingencies. This leads to group forming. Then reproduction captures the successful grouping and passes it on.

Simulation

The simulations were done on a DEC alpha PC using a two dimensional matrix of squares. Each square was either empty or filled with one agent. Multi-cellular agents also occupied only one square. This made it easier to simulate the agent moving around. An empty square can contain some amount of energy which can be harvested by an agent coming into the square. Empty squares can incrementally increase their reservoir of energy on a scheduled basis, based on a parameter which represented an environmental "sun". To begin the simulation, all the squares had an equal but arbitrary amount of energy and an arbitrary number of single cell agents were created.

Each agent was initially composed of a single cell with three abilities which included harvesting, defending and attacking with varying degrees of potency. To represent some conservation of functionality, the sum of the strengths of abilities was equal to a constant. This means that if an agent was a great harvester it would not be a great defender or attacker, and vice versa. The ratios of the abilities was quantized into 8 categories. Each category represented an identity marker that the agent would present to other agents on contact.

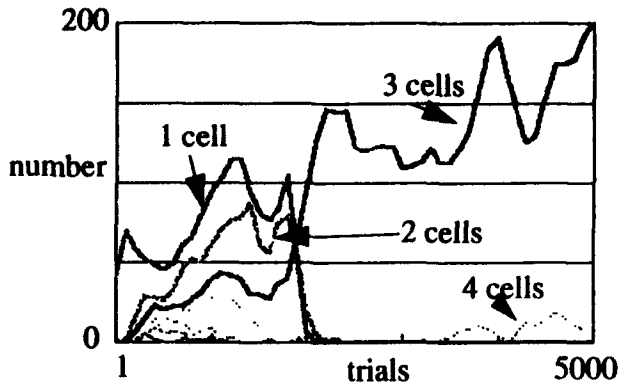

Figure 2. Plot of population of agents with different number of cells over time. The dominant group has 3 cells showing that they have an evolutionary advantage.

The following parameters were used in the simulation:

size of environment =30X40; background energy = 10 units every 10 cycles
lifetime = 100 cycles; initial energy = 100 units; reproduction energy = 500 units
number of initial agents = 40; harvest efficiency = 10%; sense range = +- 10 squares
cost of existing = 1 unit; cost of moving = 1 unit; cost of defending = 10% of self-energy
cost of failed attack = 1/2 of the agent's energy; gain of good attack= energy of loser

With no learning, agents moved around at random. With learning enabled, agents began forming clumped collections. These clumps dynamically formed in one area and then reformed elsewhere, presumably based on how energy flowed across the clumps. After 5000 trials, agents with 3 cells became the dominant group as shown in Figure 2. This run shows that multicell agents have an evolutionary advantage over single cell agents, with the current choice of parameters. More experiments need to be done , to determine if the assumptions used for this run reflect underlying tradeoffs in the real world.

Conclusions

The driving force of behavior and evolution in this system is a self-interest function. Self interest and a primitive anticipation mechanism lead to group formation, where self-interest is more easily satisfied and with greater stability. This phenomenon may be viewed as synergetic or catalytic. The simulation showed that when the agents were run with certain cost-benefit transactions they tended to reproduce better if they had more than one cell. On the other side, the increasing costs of increasing the number of cells indefinitely inhibits reproduction with too many cells. Future experiments will examine how form and function are affected by multi-cell agents.

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Evolutionary change at various levels in artificial organisms

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Organisms can be described at various levels (molecules, cells, tissues, organs, the whole organism, collectivities of organisms) and change that occurs at one level is complexly related to change at other levels. Recently artificial life methods have been proposed for understanding organisms and how they change. However, because of the complexities of any particular level, simulations tend to concentrate on one or two levels of organization, thereby missing the imporant interactions among levels. For example, neural networks concentrate on the neural level. Change can be analyzed at the neural level (e.g. change in weights due to learning) and at the performance level (e.g. change in the error rate in the learning task). Genetic algorithms concentrate on the fitness level, i.e. on how the fitness of a population of entitites changes across successive generations.

This paper describes simulations of evolving populations of artificial organisms that are simultaneously described and analyzed at four levels: genetic, neural, behavior, and fitness. (Cf. Miglino, Nolfi, and Parisi, in press; Nolfi and Parisi, in press) The organisms live and behave in an environment with specific properties (ecological neural networks; Parisi, Cecconi, and Nolfi, 1990) and reproduce on the basis of their fitness. A neural network simulates the nervous system of each organism and controls its behavior in the environment. What is inherited by the offspring of the reproducing individuals is a genotype which maps in complex ways to the phenotypic network (development; cf. Belew, 1993). Hence, each individual can be described and compared with other individuals at four levels: (a) its inherited genotype, (b) the neural network resulting from execution of the developmental instructions

constituting the genotype, (c) the behavior (input/output mapping) resulting from the interactions of the neural network with the particular environment, and (d) the fitness of the individual.

At all levels except the fitness level it is possible to distinguish between a functional and a nonfunctional component. At the genetic level some genetic information can remain 'dormant' and do not influence the resulting phenotypic network. At the neural level, some neural structure (neurons and axonal branchings) can be generated during development but fail to become part of the functional network controlling the individual's behavior. At the behavioral level the individual may be theoretically capable to respond to some sensory inputs but fail to actually experience those inputs during its life; hence, the individual's fitness is determined only by its actual experience and behavior.

If one compares evolutionary change (i.e. change from one generation to the next) at each of the four levels, the following results are obtained.

First, there is more change at the lower levels of the hierarchy than at the higher levels. There is a lot of change at the genetic level which is not expressed as change at the neural level. Much change at the neural level does not translate into behavioral change and much behavioral change does not translate into change in fitness. Since adaptive change is defined as change that arrives at the fitness level and is selected because of its improved fitness value, much change that occurs at lower levels but does not translate into fitness change, appears to be adaptively neutral.

Second, although neutral change may not affect higher levels in the particular organism it can have significant effects for the population. The locking in of much change at the lower levels and its invisibility at higher levels has the consequence that at higher levels there may be long periods of stasis (no change) followed by sudden significant changes (punctuated equilibria; Eldredge and Gould, 1972). A further small change at lower level can interact with the previously accumulated change and suddenly emerge at higher levels.

Third, change at higher levels may be dependent on pre-adaptations (or exaptations, Gould, 1991). Structures that have emerged in previous generations for chance reasons or because they were adapted for a certain function, because of a further small change may suddenly turn out to be adapted for a new function. The new structure for the new function would not have emerged in the absence of the previous pre-adapted (or not-adapted) structure.

The four levels examined in these simulations (genetic, neural, behavior, and fitness) do not exhaust the levels that can be recognized in biological-behavioral systems. For example, individual organisms interact with other organisms, directly or through the environment, and these interactions can collectively determine a further, supraorganismic, level of organization that may influence and be influenced by the lower, organismic, levels. The results of some simulations that explore this further level will be analyzed and discussed.

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