Organodynamics: A General Theory of Dynamical Systems based on Chance Organization

Part V of V: Autocoorganization

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Autocoorganization

Autocoorganization can be understood as the “dynamics of organodynamics”.

In previous articles, I have said a number of things about the dynamics of organodynamics. I have indicated that it is stochastic, and that it is therefore described using probability and information theories. I have also said that how the organization of organodynamic systems changes over time drives the dynamics. I have even gone so far as to say that the stochastic change over time within an organodynamic system is a piecewise time-homogeneous pattern, and that its complexity is such that it can form an organodynamic web.

However, I have not yet described all I need to in order to account for how it can manage its own behavior in a manner that promotes its continued persistence over time. Although I have demonstrated how its stochastic regulation can prevent it from reeling chaotically out of control, I have not yet shown any mechanism based on probability alone that permits it to adapt its behavior when necessary in order to promote its continued existence.

The specification of those dynamics is the responsibility of one of the seven organizing principles of organic complex systems. Specifically, the specification of the organizational dynamics of organodynamics is the property that we have named autocoorganization. The purpose of this article is to present these ideas.

Unpredictability in Complex Dynamical Systems

Predictability and prediction are conveniences insisted upon by scientists and engineers. But highly complex systems, such as life on earth, don’t really care about the consistencies associated with predictability. Complex systems such as life on earth are sometimes predictable and sometimes not. Yet life persists. It persists sometime through regularity of behavior. But at other times it exists because it can suddenly alter its behavior in drastic ways – and adapt to changes in its environment.

This is a stunning achievement for a stochastic system. Yet it is observed nevertheless. The question is “Can a dynamical system such as life on earth exhibit this striking adaptive behavior in a manner that, while going beyond me predictability, can be described on the basis of chance alone?”

Geneticist Jacques Monod, Nobel Laureate and early investigator into gene expression and gene regulation, observed [Monod 1972]:

> The thesis I shall present in this book is that the biosphere does not contain a predictable class of objects or of events but contains a particular occurrence, compatible indeed with first principles, but not deducible from those principles and therefore essentially unpredictable.

It just may be that some complex systems in nature simply do not admit to predictability. Would this eventuality "put science out of business" in those areas? Can science tread were predictability is not an admissible trait? The answers to these questions are for scientists to decide. I expect that engineers and mathematicians, however, will not be daunted by this prospect.

A disrupting aspect of these types of complex systems – particularly life on earth - is that they interdependently create each other, and themselves. This is a less-than-tidy trait that frustrates predictability. We have, in Part II, called this self-creational concept, as articulated by organodynamics, autocoorganization.

Not only do these systems co-create each other, but also they repair, maintain and generally manage each other. In general, then, we shall say that they organize each other – which includes all of these other "management" activities. Thus rather than use the term autocogeneration, we shall use the more inclusive term autocoorganization.

But this untidiness does not end there. These systems seem also to regulate each other and themselves, through and by themselves. In the view of organodynamics, there are of the particular organizations to which there is an affinity, and also certain organizations to which there is an aversion. This is organizational regulation.

Even beyond that, it happens that certain sub-organizations (topological subspaces, or sub-topologies) influence other sub-organizations. This is organizational adaptation. Thus, some of these types of systems create, organize and regulate themselves – and then adapt to changing environments (super-systems).

In computer science and engineering, we call these activities "operational management". That is, autocoorganization is essentially self-management. These are, of course, very complex behaviors that may disrupt predictability to achieve persistence. This is the nature of adaptation.

The Mahayana Buddhists apply a phase to describe the nature of the universe that comes close to capturing these kinds of "dynamics". The phrase is "interdependent co-origination". (Many physicists may hesitate to use the term "dynamics" here, because for them, the term carries the connotation of predictability, if not deterministic predictability. However, for systems theorists, "dynamics" means something closer to "time evolution", whether predictable or not.)

To complexify the situation further, all of these organizational dynamics occur under the auspices of chance variation – stochasticism, statistical dynamics. These types of complex systems are appropriately modeled as dependent stochastic processes – but stochastic processes with very interesting properties, indeed. Some of these processes may approach a stationary probability distribution as a limiting distribution – as we saw I the Part IV. Some may resolve to periodic behavior. Others may diverge wildly as time evolves.

The most interesting possibility may, however, maintain types of dynamics that are self-regulating – and even self-adapting. Either of these types of behaviors would be neither stationary, nor divergent. And they may or may no be periodic. In any event,

some cybernetics-like mechanism must be present within such systems. The theory of organodynamics is responsible to investigate and characterize these behaviors.

At this point in time, I have not succeeded in completing the development of these capabilities in organodynamics. And, admittedly, these issues are at the heart of the matter of what makes these kinds of complex dynamical systems distinct from those normally studied by dynamical systems theories and mathematical physics. At this time, this investigation is at the forefront of considering how to define and to mathematically represent these kinds of time evolutions.

However, in this section, we shall briefly present my thinking at this time as to how the development of the stochastic mathematical approach can be developed that can capture what makes these systems behave the way they do.

Science may be ultimately interested in predictability. But engineering goes beyond predictability – it wants to know "how to build one of those things". Whether or not we can achieve predictability with these kinds of complex dynamical systems – a class that we have called organic complex systems, perhaps we can nevertheless develop, through a new mathematical modeling paradigm, a better understand of how they work.

Lets now look deeper into the three aspects of autocogeneration that we outlined here.

The Dynamics of Entropic Change

As a stochastic process evolves, each time step is represented by a probability space that we abbreviate as a probability distribution. Time evolution in a stochastic process can be represented as either continuous or discrete. As we have said, in organodynamics for now we are working with discrete stochastic processes only.

In the simplest of cases, the probability space remains the same for all time steps in the stochastic process. Such processes are called time-homogeneous, abbreviated to homogeneous. A good deal of stochastic process theory restricts its focus to homogeneous stochastic processes, because they are considerably more approachable – and a great deal of interesting theory can be made about them.

However, in organodynamics, we do not have the luxury of that. Right away, we must contend with nonhomogeneous stochastic processes. The reason for this is that the class of systems in which organodynamics has taken an interest are complex enough so that they are almost always nonhomogeneous – eventually.

For example, biochemical systems – which admittedly have served as the prime inspiration for the development of organodynamics – are nearly always nonhomogeneous. To see why, consider a collection of biochemical entities (molecules and macromolecules) within a living cell. The cell is semipermeable. Therefore, its environment is constantly changing – molecules are constantly entering and leaving the cell. And each change in constitution of the cell requires, from the perspective of organodynamics, a change in the sample space that is used in organodynamics to initiate the mathematical model that is used for the cell. And a change in sample space necessarily involves a change in probability distribution. And, of course, these changes are occurring, within our organodynamic model, on a time step basis within a stochastic process. Consequently, this stochastic process is nonhomogeneous.

Of course, in biochemistry, chemical entities (ions, molecules, etc.) enter and leave the cell on a relatively infrequent basis – as compared to the rate of other chemical activity

that goes on within the cell. That is, relatively speaking, a lot happens within a cell chemically between the occurrences of arrive-or-leave events. And, between those events, the constitution of the cell remains unchanged – and thus the sample space. In fact, there is often no compelling argument put forth to suggest that the probabilities of those sample points during an interval of time with the cell when nothing has left ore entered the cell.

Consequently, during those time intervals, the probability distribution for each time step within the interval stays the same – and we have, for some finite stretch of time steps, time-homogeneity. This discussion paints a picture that we have called piecewise-homogenous stochastic processes. That is, we have time-homogeneity. But it is occasionally punctuated at certain time steps by a change in probability distribution, which maintain until the next punctuation.

Entropic Functionals and Their Applications

Of course, piecewise-homogeneity is a little "tamer" than wildly fluctuating nonhomogeneity. But it is not as "stable" as pure homogeneity. We are beginning to get a picture here of a continuum of possible degrees of "wildness". That is, sometimes the probability distribution is staying the same; but at other times it changes. But how do we measure this "wildness" – this degree of certainty or uncertainty?

Of course, information theory has already shown us how to do that – via the entropic functionals. The foundation of the entropic functionals is, of course, entropy. Since entropy is a functional on a probability space (distribution), it can serve as a measure of the degree of uncertainty inherent in that distribution.

Of course, degree of uncertainty is an intentionally vague term – intentionally vague so that it has many specific applications. This fact promotes its application to many possible situations – in fact any situation that has probabilities. This immediately seen by inspecting the definition of entropy:

$$H(X) = -\sum_{i=1}^{n} p(x_i) log(p(x_i))$$

Clearly, the definition of entropy: 1) requires probabilities as inputs; 2) requires probabilities for each sample point in the probability space as inputs; and 3) requires no inputs other than probabilities.

And the range of allowable interpretations of the concept of "uncertainty" is given broad latitude, indeed. Such interpretation could be a "good" trait, such as "freedom" or "opportunity". Or it could be a "bad" trait, such as "instability", "unpredictable" or "chaotic". Of course, as a piece of mathematics, it has no intrinsic moral value – only the potential for moral interpretation.

However, there is some consistent portent that can be attributed to the value that entropy assigns to probability distribution. It is that as entropy increases, so does uncertainty – or whatever application the concept of uncertainty is being put to in any given situation.

Moreover, entropy is increased by altering a probability space in either of two ways: 1) increasing the size of the sample space, and 2) "leveling" the probability assignments

of the sample point – by changing so that they are more equally-likely. Moreover, doing the opposite decreases entropy.

But information theory does not stop with this single functional. Rather, it defines an arsenal of other functionals that are based on entropy. In general, these are all functions of entropy – and share the three traits listed above.

Essentially, the other entropic functionals serve to broaden the range of applications of the entropic concept. For example a number of these functionals are defined to operate on joint distributions, and their applications center around measuring the degree of statistical dependence or independence of these joint distributions.

It turns out that there is also some consistent portent regarding the increase in statistical dependency between two joint probability (or chance) variables. It is that, as the degree of dependency decreases, so does uncertainty.

One application of this fact is to define the two chance variables involved so that they represent consecutive time steps in a stochastic process. In this application, then, "decreased uncertainty" can be reasonably interpreted as "increased predictability"; and "increased uncertainty" can be reasonably interpreted as "increased unpredictability".

The Entropy Process

This discussion gives rise to a possible approach to characterizing the time evolution, the dynamics, of an organodynamic stochastic process (OSP). It is to associate with each time step of the OSP the entropy of the probability distribution (OPD) for that time step.

If this were calculated for each time step of the OSP, then we have a new process (or sequence) whose time step values are the entropies of the corresponding time steps of the initial OPD.

This new process is a deterministic sequence of real numbers that we shall call an entropy process. Such an entropic process serves as a characterization of some kind of "uncertainty behavior of the initial OPD over time. This sequence could exhibit any kind of behavior: asymptotic, divergent, periodic, aperiodic, etc.

As we said above, it is up to the investigator who is constructing such a model to decide how entropy is to be interpreted for the application at hand. Is entropy to be interpreted as instability, inaccuracy, opportunity, freedom, or some other dynamic? As is always the case for applying mathematic, the modeler must make these choices.

Other Entropic Processes

The idea of an entropy process can be generalized to include the other entropic functionals beyond entropy. These shall generally be called entropic processes. They are all deterministic, real-valued sequences that characterize the degree of uncertainty of an associated organodynamic stochastic process (OSP) in some manner or other – according the particular interpretation of entropy for the application at hand.

If the chosen entropic functional is, for example, joint entropy, then the entropic process is referred to as the joint entropic process. If it is mutual information, then it is referred to as the mutual information process.

Notice that certain of these entropic processes (e.g. a joint entropic process) characterize the "uncertainty" of a single probability distribution. On the other hand, others of these entropic processes (e.g. conditional entropy, mutual information) characterize the degree of dependency, or perhaps of predictability (if time is involved among the chance variables).

On the other hand, if relative entropy is the entropic functional that is involved, then the characterization is determined by the entropies of the two parameter probability distributions.

In any event, the use of entropic functionals to define these associated entropic processes provides a broad range of applications of information theory to the characterization of the time evolution of complex dynamical systems.

The Dynamics of Autocoorganization

Autocoorganization provides a set of mathematical mechanisms that can describe how the organization of an organic complex system changes over time.

It has already been pointed out that organodynamics posits that the change in organization over time within a certain class of dynamical complex systems is subject to chance variation.

However, there are propensities at work within organodynamics that typically constrain these organizational changes so that they are not generally wild and unconstrained. These propensities are defined by, and peculiar to, the class of applications targeted by organodynamics. Organodynamics identifies their common nature, as well as outlining their mathematical properties.

These systems are generally self-creating (autopoietic, self maintaining, self–regulating and adaptive. The encompassing term that we use in a way that accounts for all of these traits is self-organizing. Collective, organodynamics calls these properties autocoorganization.

But, how can a system create itself? Must it not pre-exists itself in order to take the action of creating itself? The resolution of this apparent paradox is that, being a system, it is comprised of components. And, some of these components do pre-exist others, and so can create the others. This, of course, is precisely what occurs in embryo development (ontogenesis) – or in the lifespan of any living organism. In fact, this autopoiesis is occurring throughout the biological world at every level of organization, from cell to ecosystem [Maturana 1974].

Once the system arises whole from this transformation, then the same components can maintain and regulate each other, and potentially also change their interrelationships (or component mix) in order to change the relationship of the system to it environment. This type of change is, of course, adaptation.

For these reasons, the appellation autocoorganization seems apt.

Organodynamics attempts a mathematical description for these dynamics. Being a general theory of dynamical systems, organodynamics makes no attempt to "show cause" or provide explanations for these dynamics. Rather, explanations of causality are left to the individual applications of the theory (the specific members of the targeted class of complex systems).

However, it is the responsibility of organodynamics to provide a formal, mathematical, mechanism for describing these behaviors over the time evolution of these systems. As we have discussed in earlier articles, organodynamics uses the constructs of probability theory and of its offshoots information theory and the theory of discrete stochastic process to provide these descriptions of these dynamics.

This mathematics has already been presented in the earlier articles of this series. However, in the remainder of this article, we shall delve deeper into these matters and discuss how organodynamics uses mathematics to describe the various aspects of autocoorganization that we just outlined. We shall also inspect a couple of important exemplar complex dynamical systems from molecular biology and see how this mathematics plays out in those domains.

As it has developed, the world of modern biology has been slow to broadly adopt a general understanding of the foundational and significant role of chance variation, stochastic processes and the entropic functionals of information theory in biological systems. However, I shall cite a few renegade biochemists, though historically established and renowned (such as Oparin and Monod), to support the case for a fundamental and profound role of stochasticity in biology.

On the other hand, there has been a recent spate of articles published in established science journals that may signal a turnaround in the realization of a significant role of stochasticism in the life science – especially in molecular biology. Near the end of this article, we shall survey some of this research and relate it to autocoorganization and organodynamics.

Stochastic Self-generation and Persistence (Autopoiesis)

It is apparent that the entities that comprise biological organisms create and organize each other. Says Jacque Monod:

> The organism is a self-constructing machine. Its macroscopic structure is not imposed upon it by outside forces. It shapes itself autonomously by dint of constructive internal interactions…. the constructive interactions are microscopic and molecular…. [Monod 1972, p. 46]

Monod proceeds to explain that biosynthesis is the one invariant common within the structure and functioning of all living beings:

1. In its structure: all living beings, without exception, are made up of the same two principle classes of macromolecular components: proteins and nucleic acids. What's more, these macromolecules are in all living beings constituted by the assembling of the same residues, finite in number: twenty amino acids for the proteins and four kinds of nucleotides for the amino acids.
2. In its functioning: the same reactions, or rather the sequences of reactions, are used in all organisms for the essential chemical operations: the mobilization and storing of chemical potential, the biosynthesis of cellular components. [Monod 1972, pp. 102-103].

Within a biological organism, as within a cell of a biological organism, the parts co-create each other, from which in turn emerges the whole. So, too, within a species, the organisms co-create each other. This is self-generation. It is adequately described as co-origination. And, since it involves an entity creating itself by virtue of its components organizing themselves into a particular set of interrelationships, I shall name this mechanism autocoorganization.

However, there is no systemic reason why the property of self-generation, or autopoiesis must be limited to biological systems. It may be that we have no experience with any self-generating systems that are not biological. But that observation does not rule out their potential existence. From a purely systems-theoretical perspective, there is no reason to rule out that possibility.

There is no reason to restrict autocoorganization to biological systems. In fact, our definition of organic complex systems need not exclude systems other than the biological – whether or not we can at this time cite examples.

Autopoiesis

In the nineteen-seventies, neuroscientist Humberto Maturana proposed a theory of organization of biological systems that he named autopoiesis. This theory is one of the first formal articulations of the self-generating aspects of biological systems. Maturana proposed this principle as fundamental to all living systems.

Maturana describes autopoiesis in a 1974 article:

> …[A]utonomy in living systems is a feature of self-production (autopoiesis), and… a living system is properly characterized only as a network of processes of production of components that is, continuously and recursively, generated and realized as a concrete entity (unity) in the physical space as the interactions of the same components that it produces as such a network. This organization I call the autopoietic organization, and any system that exhibits it is an autopoietic system in the space in which its components exist; in this sense living systems are autopoietic systems in the physical space. [Maturana 1974, first page of article]

We propose this same property, autopoiesis, as an essential element of autocoorganization. Essentially, we propose autocoorganization to begin with the ideas of autopoiesis, but to extend them to go beyond origination and to include self-maintenance, self-regulation and self-organization.

Stochastic Autopoiesis

The interactions among the molecular components that are involved in molecular biology are the activities of diffusion processes – processes that also involve both affinity and aversion forces. In other words, they involve varying degrees of randomness (chance variation) while at the same time are subject to conditional dependences (forces of nature) that constrain that randomness.

These dynamics can be observed to be at work within the process of protein synthesis, which is a particular example of autopoiesis – component entities co-creating and regulating each other.

This exemplifies how the autopoietic processes of molecular biology exhibit stochasticism.

Parallel Processing

A number of the concepts that we present below require the notion of the simultaneous operation of two or more organodynamics stochastic processes (OSPs) and organodynamics dependent stochastic processes (ODSPs).

I introduced such an idea in Part III. There, I discussed the notion of the "forking" of ODSPs at some specific time step into multiple "parallel operations", wherein the reorganization that occurred at that time step would be partitioned across these multiple processes. We also discussed the complementary operation of "joining" multiple ODSPs into a single ODSP at some time step, wherein the reorganization of components that occurred at that time step would be the result of some kind of combining of the respective organization of the separate ODSPs previously.

When these ideas of "forking" and "joining" these various ODSPs are combined, the notion of an organodynamic web can be defined. We did not fully develop these ideas in Part III, but rather referred to another source [HollandJG-II 2011] in which these ideas have been more fully developed.

Nevertheless, we shall have occasion to build upon these ideas below. Hopefully, they have been represented here and in Part III in a manner that is sufficient to motivate any new ideas put forth in the present article.

The Complexity of Resilience and Adaptability in Natural Systems

Highly complex dynamical systems, including living systems, exhibit flexible, robust, adaptive and nondeterministic dynamics. They possess certain mechanism that resists changes in the properties of species – for example, gene replication. In other words, each species appears exhibiting some "momentum" of staying the same. Yet, at the same time, occasional change is permitted – for example genetic mutations and copy errors.

However, there is no dogma here, because species are often seen changing their nature by any means necessary "in order to remain extant". So, if there ever had been some intention to express any particular nature, biological species seem to be "willing" to abandon such "goals" at any time in order to remain extant! And, they seem "willing" to change to any other nature that is available in order to remain extant. That is, ultimately, biological species are opportunistic beyond "any scruples", or "intention" to realize some goal or "purpose". If, indeed, there is any intention or purpose involved in the evolutionary process, such intentions do not survive adaptation.

Moreover, an unimaginable compliment of distinct opportunities seems to be offered to biological species whenever organism reproduction occurs. And, very many of these types of changes seem to be realized at once. However, it typically occurs that few of these species changes survive, and therefore do not persist. However, several often do.

It is said that these survivors live to "compete" for resources against each other. However, such an analogy is probably too generous. There is no evidence that bacteria are sufficiently self-aware or sophisticated to be involved in activities such as "intending to compete". While "competition" may be a colorful analogy, it may not be involved in the actual dynamics of bacteria evolution. In the first place, "compete" ,, as often used, implies that some intentionality, purpose or goal to survive – and perhaps to survive in a certain manner - is at work within these organisms and within these dynamics. While this may be true in higher-level organisms of the animal kingdom, it is not clear that bacteria have any such teleological nature. And, there seems to be insufficient evidence that these "players" (bacteria) do, in face exhibit teleological aspects. Whether or not plants have intention is subject to debate.

If intention, purpose or things teleological were required for the enablement the dynamics of autocoorganization, then an argument over whether teleology is at work here may be worth having. However, it could also well be that some mechanism that does not require teleology can account for the dynamics of autocoorganization. I shall explore this possibility later below.

As well, the changes in situ that biological species are observed to make within the regime of species evolution are seldom radical. So species change is generally conservative and seldom "wildly chaotic".

Lets summarize, then, what appears to happen within the regime of species evolution. At any time that an organism of a species reproduces:

1. A very small percentage of the possible ways that a species can change are made available – through chance variation.
2. However, this small percentage of chance variations represents a large number of possible ways that the species can change.
3. Many of those possibilities are then realized as new versions of the species.
4. A few of those typically attrite immediately, while a few survive. Which ones survive and which do not is subject to chance. There also may be statistical dependencies at work to confer certain advantages of survival to certain of these manifestations, but these are not certainties.
5. If the conditions of the environment have not changed, then the chance of organisms with the pre-existing traits surviving may be elevated. In any event, the conditions of the environment influence the chances (conditional probability, statistical dependence) of each of the possible realizations of the species surviving.
6. In any event, it seems that there has been a propensity for the existing version of the species to most often not attrite. This eventuality is analogous to regulation in cybernetics, where changes in state are not allowed to be permanent. Rather, the system is reconstituted to it original state after change was attempted.
7. However, occasionally, the existing version of the species does attrite, while altered versions of the species survive. This is called adaptation in evolutionary biology. This adaptation is, in a sense, the reverse of the regulation just described – because in one case the existing version of the species underwent attrition, and in the other case it persisted.
8. There is no guarantee that this dynamics is in any way "optimal", in the sense that the "best" organisms survive. This is because the dynamics immediately reward short-term survival over long-term advantage. Any long-term advantage must first survive short-term risk in order for the long-term advantage to manifest.

Overall, the behavior of evolutionary change among bacterial species over time is ultimately unpredictable. It may be periodic, aperiodic, "temporarily" asymptotic, or other patterns of dynamical organization over time. However, it is seldom wildly unpredictable. So, while it is ultimately nondeterministic, its behavior does undergo certain probabilistic constraints. There are "things that can be said about it", while at the same time remaining illusive.

In the realm of theories that seek to describe system dynamics, these traits are unusual. Clearly these types of systems are very interesting and beg to be

characterized by a theory. However, they are ultimately unpredictable, while at the same time being stochastically constrained.

I do not know of other dynamical systems theories that claim these kinds of dynamics as their domain. And I would like for organodynamics to accomplish this. And it shall do so within the systemic property of organic complex systems that it calls autocoorganization.

The Mechanisms of Autocoorganization

In this section, I shall attempt to imbue autocoorganization with a sufficient set of mechanisms so as to be able to describe the complex kinds of dynamics that were illustrated by the example of primitive biological organisms that we presented in the previous section.

I shall, then, specify that autocoorganization is a mechanism of origination and persistence that operates according to a particular set of dynamical principles. These dynamics promote persistence via the exhibition of five systemic properties. These five traits are:

- Regulation
- Diversification
- Proliferation
- Attrition
- Adaptation

The interplay of these five properties can be considered as a dynamics for organodynamics. In other words, autocoorganization can be considered to be the dynamics of organodynamics.

I have already, across these five articles, identified a number of aspects of the dynamics of organodynamics, including organizational change, stochasticity, etc. However, these five attributes of autocoorganization add considerable specificity to the nature of these dynamics.

These five properties will be recognized as providing similar functions to the various mechanisms of cybernetics. However, as we shall discuss, the mechanism of organic persistence is more general than cybernetics.

We shall now describe these properties. As we do, we shall comment on the analogs of these traits in both cybernetics and in the evolution of biological species on earth.

Regulation. Regulation is the capability to continue to same behavior for some period of time. Regulation is the ability to maintain an affinity for a particular behavior, while still permitting variations in behavior that away from that behavior for which there is an affinity. Regulation can be referred to as affinity dynamics. Regulation is similar to elasticity, in that there is a propensity to “bounce back” to some particular state for which there is an affinity. “Negative feedback” in cybernetics is a special case of affinity dynamics, or regulation. The evolution of biological species exhibits a number of regulatory propensities.

Diversification. Diversification is the capability to behave differently than any particular specific behavior for some period of time. In this sense, diversification is a contrary behavior to that of regulation. Diversification can be referred to as aversion dynamics. Essentially, diversification is a generator of variety. “Positive feedback” in cybernetics is

a special case of aversion dynamics, or diversification. The evolution of biological species exhibits a number of diversification and variegating propensities.

Proliferation. Proliferation is the concurrent or simultaneous instantiation of both regulation and diversification in a large number of occurrences. The function of proliferation is to provide ample opportunity for the availability of instances of both. Proliferation is a provider of the abundance of variety. Cybernetics has no counterpart to proliferation. The evolution of biological species exhibits the characteristic of proliferation.

Attrition. Attrition is the cessation of an entity or a behavior (dynamical systemic property) within an organic system. This may be due to a failure to appropriate critical resources, or for any number of other reasons. The attribution of causation is left to applications of organodynamics. But attrition describes the mechanism within autocoorganization by which it occurs. The analog to attrition in cybernetics is the decision apparatus within the regulator component that directs an effector to change its behavior in response to feedback. The evolution of biological species exhibits the mechanism of natural selection, which is described as “selecting” (i.e. “choosing”). However, such a personification implies that there is some “intension” on the part of some “selecting” entity. I regard this as an unnecessary attribution. I seems absolutely sufficient to employ the passive concept of attrition here, rather than the active concept of “selecting” which suggest that teleology must be at work.

Adaptation. Adaptation is a change in behavior or constitution of an organic complex system that enables it to persist for some period of time. In this sense, adaptation represents contrary behavior to that of regulation. While diversification enables such a change by offering a changed state, adaptation is the realization of such a change for some persistent period of time. The prevalence of either regulation or adaptation results from attrition. Attrition may result in the prevalence of either regulation or of adaptation. In cybernetics, adaptation is effected through positive feedback followed by a decision to select differing behavior from the immediate past. Biological systems exhibit an instance of cybernetics called homeostasis in which the dynamical system can undergo, at least temporarily, a change of state for some period of time. Natural selection in species evolution effects adaptation by “selecting” an alternative (e.g. a mutant) to what went before. However, while both positive feedback and natural selection are understood as “acts of selection”, attrition is passive and requires no involvement of any “actor” – although it does allow it.

Notice that, while these systems are persistent, they may not be deterministic. In fact, all five of these properties can be described via the mechanisms of probability and information theories – which we shall do in a section below.

This discussion should make a number of aspects evident regarding these dynamics. 1) These five properties interact in such a way that the persistence of these processes are both enabled and promoted without being guaranteed. 2) Each of the five properties can be described by probabilistic mechanisms alone. 3) While the time evolution of a process may be narrowly constrained by these probabilistic dynamics, the precise states of future time steps are not determined by it. 4) A dynamical system (or process) that exhibits these dynamics is generally unpredictable, in the sense of specifying the future time evolution of the process on a step-by-step basis.

Autocoorganization First Example: Species Reproduction and Evolution

Before developing those mechanisms, however, lets look an example from a natural complex system – biological life. While these mechanisms proliferate throughout biological systems, lets look at a specific example from the foundations of cell and molecular biology: species evolution.

This mechanism is broadly understood, so we shall provide only a cursory description here – a description that emphasizes how the five properties of organic persistence that we outlined above come into play. We shall look at some detailed science a little later below; but for now it will be useful to be more conceptual and construct a "toy explanation" of the process. We shall first present a highly idealized model, and then discuss how each of the above five properties figure into the general scheme.

With apologies to any geneticists among the readers, please allow this humble systems theorist offer a simplistic explanation of gene copying in cellular reproduction….

We shall present the case of the simplest of cell types – prokaryotes. For more complex cells, including eukaryotes and especially germ cells in sexual organisms, the explanation is considerably more elaborate. Nevertheless, the five properties of organic persistence maintains in all of these cases. Thus, a discussion of the simpler prokaryotes will suffice.

The DNA replication process begins when certain initiator proteins locate certain origin position within the DNA double helix. These proteins unzip the double helix at these points and separate the molecule into its two strands. At this point, any of a number of proteins of the complex DNA polymerase attach to each of the two strands and initiate the copy process. Each of these strands is the complement of the other. These proteins then proceed to create complements of each of the initial two strands. Subsequently, each initial strand forms a new double helix by intertwining with the complement that it created. The result is two new double helix instances, each of which is an exact copy (almost always) of the initial one. Moreover, each of the two strands of the initial DNA instance is a member of one of the two new molecules; and, of course, each of the new molecules contains one original strand and one newly constructed strand.

This replication process, however, is not 100% perfect – although it is nearly so. This level of perfection is achieved by a number of "quality control" mechanisms that ensure such a high rate of success. In fact, the success rate is phenomenal – roughly on the order of one mistake for every 1 billion nucleotides copied. In addition, these nucleotides can suffer spontaneous change or damage though mutations during their life after replication. But these, also, are very rare – typically occurring in less than one-half of one percent of DNA molecules. These errors, in either case, can only be understood as random events, regardless of their rarity.

However, as rare as they are, these random alterations in DNA molecules have been essential to the evolution of biological life on earth. They are necessary and essential to biodiversity and to the continued persistence of life on the planet. If not for their occurrence there would have been essentially only one species in the history of life – and only one configuration of DNA molecule to replicate. And it is unlikely that such a species could have persisted for long. Having no ability to change and adapt, it would not have been able to survive under the changing environment of the early – or even late – earth. Thus, these "copy errors" and mutations have been absolutely essential to life on earth. Without them there would have been no evolution.

Lets again call upon Jacques Monod characterize the random nature of DNA replication – and the absolute necessity of it being so….

> We call these events [“noise” in DNA replication] accidental; we say that they are random occurrences. And since they constitute the only possible source of modifications in the generic text, itself the sole repository of the organism’s hereditary structures, it necessarily follows that chance alone is at the source of every innovation, of all creation in the biosphere. Pure chance, absolutely free but blind, at the very root of the stupendous edifice of evolution: this central concept of modern biology is no longer one among other possible or even conceivable hypotheses. It is today the sole conceivable hypothesis, the only one that squares with observed and tested fact. And nothing warrants the supposition – or the hope – that on this score our position is likely ever to be revised. [Monod 1974, pp. 112-113]

And Monod continues….

> And so one may say that the same source of fortuitous perturbation, or “noise”, which in a nonliving (i.e. nonreplicative) system would lead little by little to the disintegration of all structure, is the progenitor of evolution in the biosphere and accounts for its unrestricted liberty of creation, thanks to the replicative structure of DNA: that registry of chance, that tone-deaf conservatory where the noise is preserved along with the music. [Monod 1974, pp. 116-117]

Autocoorganization and DNA Replication

Lets evaluate our DNA regulation example to see how it supports the elements of autocoorganization that we listed above: regulation, diversification, proliferation, attrition and adaptation.

Above, we defined regulation as the ability to continue behaving the same. Clearly the DNA replication regime is a mechanism whose function is precisely that. As we have shown, the DNA replication process in life on earth is phenomenally successful in providing an extremely high probability that its copies of a DNA molecule are precise, and that the organisms that result from them will continue to behave in the same manner, and to exhibit many same systemic properties, as its predecessor.

However, the DNA replication process does permit a limited amount of diversification. This results from the rare copy errors of the replication process, as well as the rare mutation errors that occur within DNA molecules prior to the time when they are also replicated. However, regardless of their relative rarity, there are still large numbers of occurrences of these “errors” so that diversion occurs often enough to have provided considerable diversity among the population of species of life on earth over time. Of course, the levels of diversity of species has ebbed and flowed over time.

The occurrence of both regulation (continuity) and diversification of species has continued concurrently or simultaneously over time. This is proliferation.

There is the possibility that all species – the ones resulting from regulation as well as the ones resulting from diversification – persist. However, historically the natural environment, including these species, has not been able to provide sufficient resources to maintain the persistence of all such species. Consequently, as pointed out by Darwin [Darwin 1859], some of them cannot and have not survived (persisted).

There is no single consistent reason (such as being the “most fit”) why some survived and not others. Rather, the “reasons” are multifarious and complex. The most consistent observation that can be made is simply that some species ceased to persist. This is the trait that we call attrition. Evolutionary biology attributes this trait to an “act of selection” that it calls natural selection.

However, terminology “selection” implies that there is some sentient agent at work that makes some decision, or selection, of some species over others. Unfortunately, such an intellectual construct requires the identification of such a selecting, or deciding, agent. It does not seem to organodynamics that such an agent is evident or required.

On the other hand, there seems to be no requirement to assume that such a deciding agent needs to exist. Some such agent may exist, or it may not. Whether or not such an agent exists is irrelevant because a passive condition can have the same result. That is, a lack of resources can accomplish the same result as a sentient “selector”. Thus, Organodynamics calls this condition attrition. It is sufficient to simply observe that some number of species failed to survive while others did. That is, some species persists while others do not.

Attrition may result in the continued persistent of extant species. On the other hand, under certain conditions – for example random changes in the environment – existing species may be retired before novel ones. In this case, we would have the result that we described above as “change in the prevailing behavioral properties of a dynamical system for some period of time”. We call this trait adaptation.

Entropic Description of Autocoorganization in DNA Replication

It is the position of this article that autocoorganization is completely describable using various aspects of probability and information theories alone. Lets discuss this in terms of the five traits of autocoorganization and how they are manifest in prokaryotic DNA replication as before.

- Regulation
- Diversification
- Proliferation
- Attrition
- Adaptation

Regulation

We defined regulation as “the capability to continue to same behavior for some period of time”. Given this definition, regulation in DNA replication can be represented as time-homogenous Markov chain whose self-referential probability is near 1 – on the order of 10^{-9}. In the simplest possible representation of this phenomenon, the marginal sample spaces of the transition matrix for such a Markov chain could be binary, with one sample point being the current DNA configuration of the organism, and the other sample point representing all other possible DNA configurations.

Of course, many other refinements to this basic model are possible and reasonable. Which one is chosen depends upon one desire for fidelity and tolerance for complexity. Let me now discuss a few possibilities for these refinements.

One approach to embellishing this basic model of regulation is to refine one choice of Markov transition matrix sample space representation – in both margins. As described above, the sample space is defined by partitioning the set of all possible DNA configurations into two compartments: one containing the “present” DNA configuration, and the other containing all other possible DNA configuration for the current species under consideration.

There are many ways that one can refine these marginal sample spaces, and still preserve invariance of the sample space over time (which is necessary to preserve

time-homogeneity). One approach to this kind of refinement would involve deciding in advance which other DNA configurations one would allow that would still be considered to be within the acceptable boundaries of the same species as the "current" one. Once decided, categories for each could be added to the sample space. Of course, there must be a category to represent all other DNA configurations that are outside the boundary for what is considered to be the "same species".

Clearly, there are a vast number of ways that one could partition the sample space of all possible DNA configurations, each of which would be a reasonable stochastic model of the dynamics of DNA replication.

Beyond refining the sample space of the Markov transition matrix, there are many other ways to refine the initial (simplistic) Markov model described above. Because of space and time restrictions, lets mention only one more alternative refinement: the introduction of non-homogeneity.

We have mentioned in previous articles of this series that the expectation of organodynamics is that the stochastic processes used by the theory are piecewise homogeneous. This means that the probability distribution of contiguous time steps within a stochastic process will remain the same for a while, and then – at some time point – change to another distribution. For example, a change in the environment of an organodynamic process would be an example situation in which the probability distribution that "governs" the dynamics might change. Once changed, the same probability distribution might stay the same for a while, and then change again. This is the idea behind piecewise homogeneous stochastic processes.

In our DNA replication example, a number of environmental factors within a cell could result in our Markov model requiring a change in the probabilities within the Markov matrix at various points in time. Any changes in the molecular constitution of the cell, for example, could force this. Since prokaryotic cells are semi-permeable, this change could occur anytime atomic or molecular constituents enter or leave the cell.

Another possibility is that the process, while remaining stochastic and dependent, is not Markov. That is, the probabilities of the next time step depend on the realized outcomes of the past as well as the present time step.

The abilities of dependent, and independent, stochastic processes to model a vast universe of behaviors of autocoorganizational dynamics are resplendent. It is hoped that the suggestions discussed here are insightful in that regard.

Diversification

The diversification that is present in the DNA replication example can also be represented by the mathematical articulation presented in the previous section on regulation.

This being the case, then, what distinguishes such a stochastic process model that represents regulation from one that represents diversification?

The answer comes from information theory! A model instance that represents diversification exhibits higher degrees of statistical entropy than do model instances that represent regulation.

Before elaborating on this, let me first point out that this means that – because entropy is defined on a continuum of real numbered values - there is a continuum between regulation and diversification in this regard.

But which entropic functionals are appropriate to represent to degree to which a particular model instance (as described in the previous section) represents either regulation or diversification?

For now, lets limit this discussion to which of the entropic functionals that we have discussed in earlier articles of this series can be applied to these characterizations. The answer is: essentially all of them. These include: joint entropy, conditional entropy, relative entropy, mutual information and entropy rate.

Each of these can enjoy a range of applications to our model. And a complete discussion of these is beyond the scope of these articles. However, we shall mention a few examples.

First, recall that the value of the entropic functionals generally increases through the interplay of three phenomena regarding probability distributions:

1. An increase in the size of the sample space (number of sample points).
2. A “leveling” of the distribution of probabilities toward the equiprobable.
3. An increase in the degree of statistical independence between two joint chance variables.

Second, generally speaking, an increase in entropic functional values represents a higher degree of diversification and a lower degree of regulation, and conversely.

(An exception to what was just said is the functional mutual information, which intentionally measures the degree of statistical dependence. Therefore, mutual information measures smaller as statistical independence increases, and conversely. In addition, relative entropy measures the “nearness” of two specified probability distributions that are defined on the same sample space. Therefore, whether the value of relative entropy represents more of less regulation or diversification depends on the two probability distributions being compared.)

Proliferation

The fidelity of probability theory, as well as its offshoot information theory depends upon a set of theorems in probability theory that are collectively known as “the law of large numbers”. These theorems generally assert that as the number of trials of a random process increases, the actual probability values approach their expected values.

The trait named proliferation articulated above essentially specifies that, within the systems represented by organodynamics, the assumptions of the law of large numbers holds. In other words, the fidelity of organodynamics depends upon it dealing with large sample spaces – the of extended topologies. Thus, the application of probability and information theories is appropriate to organodynamics.

Attrition

Attrition is the cessation of an entity or a behavior (dynamical systemic property) within an organic system. This may be due to a failure to appropriate critical resources, or for any number of other reasons. Autocoorganization does not proffer or show cause. As a mathematical theory it provides a descriptive mechanism.

In autocoorganization, attrition is the mechanism by which the realization of certain organizations of the elements of an underling system occurs. Within organodynamics, this realization, or not, is represented via probability spaces and their distributions.

Being represented by a probability space, the mechanism by which realization either occurs or does not occur is not represented. This is intentional on the part of organodynamics. Leaving such a mechanism undefined results in a broader application of the theory. Since the underlying mechanism of realization is not specified, then the ability of this theory to describe a broad range of applications – regardless of their mechanism of realization – is assured.

This, of course, is also the case with probability theory in general. Probability theory does not specify any mechanisms of realization, and consequently works with all of them.

For this reason, the concept of attrition is essentially a "placeholder" form of realization. It simply states that "something is not realized", and does not specify "why" it is not realized. Of course, even for mechanisms that elaborate the reasons for realization or for non-realization, attrition still applies. Thus, attrition is a very general (essentially non-committal) kind of "realization dynamics".

A number of aspects of probability and information theories can be recruited to characterize these kinds of phenomena. For example, the theory of absorbing Markov chains can be used to model attrition, since attrition narrows the future possibilities of a specified Markov chain to a single outcome – attrition.

Another way to model attrition is to understand it as the elimination of a sample point from the sample space. This can be modeled as a change in sample space, and therefore a termination of the homogeneity of the stochastic process. Such an event represents a fork in the organodynamic model of the piecewise-homogeneous stochastic process.

Of course, a reduction in the size of a sample space is indicated by a reduction in entropy, assuming that all other probabilities remain proportional. This eventuality opens a discussion of the dynamics of the entropic functional values of the time steps of these stochastic processes. In any event, the behavior of various entropic functionals over time as applied to these stochastic processes offer many opportunities to characterize the behaviors of stochastic processes that are representing attrition processes.

Adaptation

Adaptation is a change in behavior or constitution of that persists for some period of time. In this sense, adaptation represents contrary behavior to that of regulation. While diversification enables such a change by offering a changed state, adaptation is the realization of such a change for some persistent period of time.

Thus, what was already said about diversification and probability theory above also applies to adaptation. Essentially, diversification is the enabler of adaptation. This means that adaptation is an outcome that is statistically dependent on diversification.

As a consequence, the interrelationship between the two is incorporated within dependent stochastic processes (e.g. Markov processes) that represent the dependency of events that involve adaptation being statistically dependent on events that involve diversification.

Several of the entropic functionals can be brought to bear to measure the degree of stochastic dependence between and among these events. These include conditional entropy, relative entropy, mutual information and entropy rate.

Autocoorganization Second Example: Protein Synthesis

Proteins are the workers of biological metabolism, which in turn includes the general processes of chemical change within biological organisms. Consequently, the creation, regulation, delivery and operation of proteins are instrumental in all living processes.

Protein synthesis is the name given to the family of chemical processes that construct all proteins. Biologists also use the phrase “gene expression”.

Protein synthesis involves several major steps. The first is the reading of the proper sequence of genes within the DNA of the cell in order to obtain the instructions for the constitution of the protein. Next, a message containing this information (an mRNA molecule) is constructed (by another protein called RNA synthetase). This “message” contains a translation of the instructions for building the protein with the correct constituents. This message makes its way (via diffusion) to a “factory molecule” (a ribosome) that reads it in order to construct a protein molecule.

A question that arises from this explanation is “How is the right number of protein molecules for any particular function maintained within the cell and the organism?” The answer is that there are certain proteins, called “transcription factors”, that can physically attach themselves to the beginning of a specific gene, and prevent (or enable) that gene from being read by the RNA synthetase protein.

In other words, the production rates of specific types of protein can be regulated by the increased production of transcription factors. However, transcription factors are themselves proteins, and are therefore produced by the same process that they are regulating”. In other words, the entire mechanism of protein synthesis is self-regulating.

The reader will recall that self-regulation is one of the five traits of autocoorganization. This fact opens the possibility that protein synthesis, like DNA replication, may satisfy the other four traits. In fact, it does. We shall discuss how next.

It should not be a surprise to the reader by this time that the entire autocoorganizational process of protein synthesis is completely imbued with randomness – to some degree or other at every step; and is therefore subject to chance variation, and can be described by probability theory, information theory and stochastic processes. For one thing, virtually every step of these processes involves chemical diffusion within or between cells. Consequently, these are essentially diffusion processes – whose dynamics are traditionally modeled by stochastic process theory.

The stochastic nature of diffusion processes is responsible for a great deal of the chance variation at work within molecular biology in general and in the protein synthesis process in particular. Here is what some molecular biologists have to offer toward this point.

[Sanchez and Golding 2013, p. 1188]

> Within a single cell, gene expression is inherently stochastic, or random. Protein-coding genes are typically present in only one or two copies per cell. Whether a gene is transcribed at any given moment depends on the arrival, by diffusion, of multiple regulatory proteins to their designated binding sites, as well as the occurrence of multiple biochemical steps

> required for initiation of transcription. These biochemical reactions are all essentially single-molecule events and thus stochastic, resulting in substantial randomness in the production of mRNA.

[Kondev 2014, p. 32] refers to this when he says:

> Ultimately the genes and proteins that turn [genes] on and off are simply molecules diffusing within the cell's interior. How the random thermal motions of these molecules and the interactions between them lead to a bacterial cell to make a decision is an intriguing problem that has been addressed by a combination of careful quantitative experimentation and theory.

Let first look at how protein synthesis satisfies the traits of autocoorganization. Then we shall further consider how a probabilistic treatment of these issues can be presented using information theory and stochastic processes.

Autocoorganization in Protein Synthesis

We just implied that protein synthesis is self-regulating, and offered a cursory overview of the mechanism. Of course the process in a living cell is exceedingly complex, detailed and multifarious. These details are beyond the scope of these articles, so we shall not describe the general biochemical mechanism in any further detail. The reader is referred to any of the vast number of sources on the subject.

What we shall do now, however, is to look briefly at each of the five traits of organodynamics and provide examples, along with occasional citations, that argue for the protein synthesis process being a manifestation of autocoorganization.

In this section we want to go further and suggest how, within the protein synthesis process, chance variation is involved in each of these five traits. We shall see that molecular diffusion is often involved in bringing chance variation into the picture.

Regulation

Monod, who with his colleagues discovered the mechanism of protein synthesis and how it is regulated, explains the robustness of this process:

> …[S]o far as regulation through allosteric interaction is concerned, everything is possible…. The way in which allosteric interactions work hence permits a complete freedom in the "choice" of controls…. In a word, the very gratuitousness of these systems, giving molecular evolution a practically limitless field for exploration and experiment, enabled it to elaborate the huge network of cybernetic interconnections which makes each organism an autonomous functional unit, whose performances appear to transcend the laws of chemistry if not ignore them altogether. [Monod 1972, p. 77].
>
> Plainly enough, the functional coherence of so complex a chemical machine, which is autonomous as well, calls for a cybernetic system governing and controlling the chemical activity at numerous points. [Monod 1972, p. 45]

So the regulation of protein synthesis is a rich and robust process – a process that is self-organizing. Let's now look at this process at a somewhat high level of abstraction, and see if we can glimpse some key to its operation.

Above, we described the transcription factor mechanism as an instance of regulation. Of course, these so-called transcription factors are also proteins, and are therefore

also constructed via the same process that they are regulating. And they may or may not be the same protein type that they are regulating. If so, then they are regulating their own rates of production. If not, then the proteins are regulating each other. In fact, both of these eventualities can occur.

In either case, a complex network of regulatory interrelationships and interdependences clearly exists among the different kinds of proteins. Of course, one would imagine certain "delays" and chance variation is also involved in the speed with which regulatory proteins (transcription factors) diffuse from their point of production (in a ribosome) and arrive at their points of delivery (a gene), where they then either disable or enable the initiation of the synthesis of the protein that they regulate.

And sometimes the arrival of such a regulatory protein at the gene does not result in a binary on/off result. Rather, such a protein may cooperate with another transcription factor in some complex way that then results in a clear on/off condition. In such a case, the regulatory protein can only be said to "effect the probability" (i.e. conditional probability) of the regulatory result.

Thus, chance variation is involved in this regulatory process in many differing articulations.

Says [Levine, et. al 2013, p. 1199] on the issue of stochastic dynamics at work in cellular regulation:

> Our traditional view of cellular regulation as a largely stead-state process is ceding ground to a more dynamic picture. Evidently, cells are controlled by regulatory factors that show repetitive, pulsatile, and often stochastic dynamics even under constant conditions.

Diversification

Diversification arises in nature stochastically when stochastic processes result in probability distributions in which entropy is high. Examples of these types of distributions include ones whose probabilities are equally likely, or near equally likely.

[Kondev 2014, p. 32] explains:

> At the heart of the process that generates such diversity is the [stochastic] on/off switching of genes.

Kondev described this switching as due to "stochastic fluctuations in the expression of genes…. [Kondev 2014, p. 31].

When this occurs, so does risk aversion. The reason for this is that the occurrence of random catastrophic events is less likely to destroy the entirety of a more diverse population. Consequently, there is a higher probability that at least one member of the population will survive and persist.

Thus, the occurrence of high-entropy distributions within the stochastic processes of nature result in diversification and produce what molecular biologists are starting to name "bet hedging" strategies.

[Levine, et. al 2013, p. 1198] describes one way that diversification and differentiation can occur stochastically within the protein synthesis process during embryotic development:

> A fundamental question in development is how transcription factors control cell fate decisions. In some cases, the role of an individual transcription factor can be complex,

> promoting multiple, seemingly conflicting, cellular behaviors. Recent single-cell studies suggest that some of these transcription factors activate in a pulsative fashion and suggests that this pulsatility may function to balance their conflicting activities.

Once "selected" in this manner – or having survived attrition – the surviving species is likely to reproduce entities that replicatet its own properties for a while. Cybernetic would label this phenomenon of self-replication as "positive feedback". Autocoorganization would call it "affinity dynamics".

The role of stochasticity in the process of gene expression and protein synthesis is articulated well by [Johnston and Desplan 2014, p. 665]

> Stochastic gene expression mechanisms may be a cost-effective way to diversify the repertoire of gene fates within a tissue. Although these phenomena involve stochastic processes, this randomness is very often well-controlled, incorporating multiple steps, apparently to insure robustness. Evolution has yielded many different mechanisms to determine stochastic cell fate specification in bacteria, flies and vertebrates. As our understanding of stochastic phenomena increases, it will be interesting to see whether common ancestral strategies become apparent or whether novel stochastic genes expression mechanism arise in individual species.

Proliferation

In our discussion of diversification above, we provided an example of how, through chance variation (e.g. some catastrophic event), a diversified population was changed (reduced) to having a small number of survivors (attrition). Thus, the result is a new probability distribution with a small number of sample points (alleles) – this being a probability distribution with reduced entropy.

Without the occurrence of the catastrophic event, the diverse population had a good chance of producing another diverse population at the next time step. Thus, from a diversified population, either a more diverse population can emerge, or a less diverse population can emerge.

Likewise, a population of low diversity will most likely produce a new generation that also has low diversity. However, it may have a low probability of immediately producing a generation with a high degree of diversity – and therefore whose probability distribution exhibits a high entropy value. However, there may be a better probability that in the long run, it has a relatively high probability of producing a diverse generation with a concomitantly high entropy.

It is also evident that the degree of entropy in any one generation is stochastically dependent on the degree of entropy in the previous generation. In fact, low entropy has a higher probability of producing low entropy in near generations; and high entropy has a high probability of producing high entropy in near generations.

In other words, the dynamics of information theory permit a non-homogeneous stochastic process to present time steps whose probability distributions exhibit varying degrees of entropy.

The principle of proliferation prescribes that all, or many, of these possible outcomes are manifest most of the time: high entropy, low entropy, etc.

Attrition

The stochastic proliferation of multiple "strategies" much of the time raises a question. If many "strategies" are manifest by the protein synthesis process most of the time,

which of these conflicting strategies become operational? The answer is “The ones that perpetuate persistence.” All others are eliminated by attrition.

This works, regardless of whether there are limited resources, or not. Under the conditions of unlimited resources, attrition may not occur. Under conditions of limited resources, attrition will likely occur.

Of course, within the protein synthesis process, the entities involved are each other’s resources. Thus limitations in resources are bound to occur stochastically as these resources consume each other. Thus, attrition does occur.

Adaptation

Adaptation is a consequence of the cooperation of diversification, proliferation and attrition.

An example of the realization of adaptation is the “bet-hedging strategy”. This phenomenon is the result of a complex unfolding of events that involve diversification followed by “affinity dynamics” followed by proliferation.

[Levine, et. al 2013, p. 1197] describes a “bet-hedging strategy” found within the protein synthesis, or gene expression, process.

> In many cases slow pulses are initiated in a probabilistic manner, effectively implementing a bet-hedging strategy whereby cells randomize their individual states to adapt to certain future environmental changes.

“Bet hedging” begins with diversification, where chance variation produces distinct alleles of a protein simultaneously or concurrently. It then happens that both (or many) of these alleles, through affinity dynamics, reproduce themselves. Now there are many copies of each allele. Subsequently, a catastrophic event occurs and most alleles suffer attrition. The remaining alleles survive. This is adaptation.

[Kondev 2014, p. 36] gives an example from protein synthesis:

> Experiments …reveal the molecular nature of that “free will” and trace it to the stochastic expression of E. coli genes. One of the expressed lac genes codes for a protein… that transports lactose molecules into the cell. That protein therefore provides positive feedback in the expression of lac genes: The more lactose permease produced, the more lactose makes it into the cell, and the more likely it is that the lac promoter will be in the on state, which, in turn, leads to more expression of the lac genes….
>
> Thanks to the positive feedback, E. coli cells exist in two different states…. Stochastic fluctuations in the expression of the lac genes – fluctuations, for instance, between on and off state of the promoter – can flip the switch and turn a lactose noneater to a lactose eater…

It is interesting to note that Kondev uses the phrase “free will”. The term “will” implies intentionality and teleology. I argue that this usage is out of place within the context of this conversation, since the described phenomena has been described only in terms of chance variation, probabilities and stochastic processes – with an absence of any entities that can be said to exhibit intentionality.

This does suggest, however, that “free will” can be mimicked (analogized) by chance phenomena alone, and perhaps articulated in terms of probability theory and its offshoots – alone. We shall take up this idea below.

Entropic Description of Autocoorganization in Protein Synthesis

It should be evident that the stochastic nature of the autocoorganizational dynamics of the protein synthesis process that were outlined in the previous section can be characterized by the application of a number of the entropic functionals of information theory. These include entropy, joint entropy, conditional entropy, relative entropy, mutual information and entropy rate.

We provided examples of how these functionals can be applied to the DNA replication process above. Due to limitations of space and time in this article, we shall leave a further deliberation about how these functionals can be applied to characterize the protein synthesis process to the reader.

Autocoorganization Versus Cybernetics

Autocoorganization and cybernetics have a lot in common. For one thing, both are concerned with system organization.

Also, they both specify mechanisms that regulate dynamical systems. These mechanisms in both cases are conditional. But in cybernetics, they are conditionally deterministic. However, in autocoorganization, they have stochastic implementations and descriptions and they are conditionally probabilistic.

It is noteworthy that both cybernetics, an act of decision-making is articulated, and some choice is made. However, the mechanism for alternative realization in autocoorganization, attrition, does not require, although it does allow for, any kind of active decision or choice to be made. Attrition is subtler than that: a consequence occurs merely by the fact that it failed to occur – rather than by being actively decided upon or effected.

Another noteworthy difference between autocoorganization and cybernetics is that, in cybernetics, a component subsystem, called the regulator, is dedicated to making and effecting the “selection”. Like cybernetics, species evolution also provides a mechanism for selection – natural selection.

Also in distinction to cybernetics, neither natural selection, nor attrition, requires such a dedicated subsystem. Rather, this function is inherent in the stochastic nature of the processes themselves.

A final and significant distinction between cybernetics and autocoorganization is that purpose, intention and teleology are evident in cybernetics. But in autocoorganization, there is no need for these concepts. They are allowed to be operational within autocoorganization, but they are nowhere required.

Teleology and Autocoorganization

I have been suggesting that the complex natural processes that have presented in this article can be described in tot by appealing only to probability theory and its offshoots. And, I have cited a number of recent science papers for the purpose of supporting this outrageous position.

However, I must admit that even many of the sources cited persistently refer to these processes using anthropological – even teleological – analogies. For example, [Johnston and Desplan 2014, p. 661] refer to “decision making” and “choice” (even while both are described as stochastic); and [Kondev 2014, p. 32] refers to “bacterial decision making”. Both [Levine, et. al 2013, p. 1197] and [Kondev 2014, p. 36] refer to

stochastic differentiating mechanisms as “bet hedging” behavior, as though some intentional strategy had been hatched by these nucleotides and amino acids while they act within the limitations of diffusion dynamics. And intimation of sentience is implied by [Kondev 2014, p. 31] when he suggests “stochastic fluctuations in the expression of genes allow a cell to exhibit a kind of free will.”

On the contrary, I see no evidence, or necessity, of requiring that any purpose, intention or other teleological aspects are at work in the dynamics of autocoorganization. I see no evidence that these “strategies” are the result of intentional design, but rather that they emerge by chance from a probability space of immense numbers of possible behaviors. If there were any enabling factor in this process, it would be the laws of large numbers.

While teleology may or may not sometimes be involved (that is, autocoorganization allows it), I do not see it as being required for the progress of these processes. I see no evidence that teleology is required for them.

Rather, the entropic dynamics of probability and information theories appear to be completely sufficient to describe biological evolution, and organodynamics. These include the chance variation involved in entropy itself, as well as dependent stochastic processes, and the diversification, differentiation, and selection inherent in these probabilistic dynamics. And also some unspecified versions of the laws of large numbers must also be assumed – as well as the involvement of very large, though finite, sample spaces.

I do not deny the existence of intention or purpose in nature, but I simply do not see at work in molecular biology or in any foundational natural processes generally. Certainly, intentionality and purpose appear to arise within higher-level biological organisms. However, I regard teleological events as complex and emergent properties that arise due also to chance organization.

Therefore, my interpretation of my own theory (organodynamics) is that it describes a generational dynamics in which probability theory, information theory and the theory of discrete stochastic processes is completely sufficient to describe (I did not say “explain”) the dynamics (origination, organization, maintenance and evolution) of complex adaptive systems. While teleological phenomena can arise within organodynamics as complex emergent properties, they are not required and the certainly are not foundational.

Therefore, organodynamics (and autocoorganization) are neither teleological nor dysteleological. They admit to the existence of teleology in nature, but do not find it foundational, fundamental or necessary.

Tractability and Organodynamic Modeling

Tractability and complexity are clearly issues with organodynamics. After all, in order to model a target dynamical system using organodynamics one must first determine the set of all possible ways that the target system can be *organized* over time. Then, one must articulate each of those organizations as an *entity* in the form of an *extended topology*. Subsequently, this set of extended topologies becomes understood as the sample space of the target system; while each such extended topology is a single sample point of that sample space. These steps alone constitute an enormous task.

Next, one must assign a probability measure to each of these sample points, these extended topologies. Not only that, but there are many more complexities that must be resolved on the way to building a useful dynamical systems model of a target system using organodynamics.

These are scary propositions, and could well lead a potential practitioner to reject the theory as intractable and too complicated to be useful. These are actually reasonable comments that deserve some response on my part – which I shall offer now.

Fortunately, organodynamics is not the first systems theory to encounter such complexity problems. Statistical mechanics and quantum mechanics have both visited these troubles before.

Numbers versus Topologies

Let me now turn to ontology – the fundamental nature of "being" of the systems that I am trying to model using organodynamics. In the field of object-oriented software technology, we make an ontological distinction that has proven useful. We make a distinction between what something "is" versus what that something "has". An automobile, for example "is a" transportation vehicle. But it "has a" color. It also "has a" engine. But it is not the case that an automobile "is a" engine, nor that it "is a" color.

The "is a" relationship is considered more important and fundamental, if for no other reason, because it is more persistent than the "has a" relationship. For example, the "is a" relationship between an automobile and transportation vehicles is much more intimate than the "has a" relationship between an automobile and a color. The first thing that an object-oriented software engineer should worry about is that all of the "is a" relationships are represented in a software design. The "has a" relationships can be attended to later.

These considerations are important to organodynamics because the class of system that it seeks to faithfully model ARE ("is a" relationship) *organizations of interrelationships of components*. This is their ontology. What those systems ARE NOT ("is a" relationship) are *numbers*. Nor ARE they vectors of numbers. These systems (e.g. living systems) HAVE ("has a" relationship) *numbers* (weight, temperature, location, velocity, etc.); but it is not the case that they ARE numbers – or even vectors of numbers.

Thus, organodynamics takes the position that it is most apropos to model these complex systems for what they ARE (organizations of relationships) rather than for what they HAVE (location, velocity, momentum, etc.).

This is why organodynamics is a systems theory that is based on system state as *organization* (articulated as a topology), rather than on some notion of state that is defined as a number or a vector of numbers.

Let me elaborate. There are several highly complex systems that fundamentally ARE organizations or interrelationships among their parts. An example of this is an ion of some atom. An ion IS fundamentally a set of interrelationships among some constituent entities – protons, neutrons, electrons, etc. This is ontologically what it IS.

On the other hand, an ion is NOT a number. It HAS some numbers as attributes. For example, it has atomic weight, electrical charge, location, velocity, momentum, etc. But none of those attributes are what an ion IS. In fact, no number is what an ion IS.

Another very good example of a thing that IS a set of interrelationships among constituents is a biological cell. It also is NOT a number. It too HAS numbers. But ontologically a cell is a set of relationships – an organization of constituent entities.

Of course, if the only modeling tool that we have is only capable of representing entitles as numbers or vectors of numbers, everything may like numbers and vectors to us. Our traditional mathematical tools are good at representing "things" as numbers, or as sets (vectors) of numbers. Therefore, we fall back on the tools that we have. This reminds me of the aphorism "If you have a hammer, then all problems look like nails."

But mathematics actually gives us more than just numbers and vectors – because mathematics is about much more than numbers and vectors. Mathematics is concerned with interrelationships. In fact, mathematics is about something more general. It is about organization. In fact, topology is a branch of mathematics that is very good at characterizing the organization of relationships among things. Topology shows one approach that mathematics can take to articulating system organization. And we have leveraged that in organodynamics.

If something IS a system organization, then I would want to model it with topology, or some other aspect of mathematics that is good at representing relationships. This is precisely the approach taken by organodynamics.

Fidelity

Models, mathematical or otherwise, exist because the target systems they represent are too complex – or otherwise intellectually inaccessible – for us to understand easily. We create a model in a way that "leaves out some of the complexities of the real thing" so that we can have a simplified representation in order to begin to understand the "real thing".

However, if the model is too simplified, then some of the salient features of the target system get left out of the model. And the purpose of the model is defeated if we leave too much out.

For my purposes, using nonlinear dynamics to model biological systems "leaves too much out". It leaves out certain salient features of living systems that seem to "make them tick" – features such as their organization. Nonlinear dynamics models systems as manifolds – sets of vectors of numbers. These vectors, to me, represent features that the target systems HAVE, but not features that they ARE. The correct ontology – interrelationships and organization - is missing in a nonlinear dynamics model of living systems. But it is present from the start in organodynamics.

Another part of the ontology of biological systems – at least to me – is its chance nature, its stochasticity. Of course, this is completely missing in any nonlinear dynamics model. So, as far as I'm concerned, nonlinear dynamics can model some of the properties that biological systems HAVE, but not some of the essential ontological nature of biological systems.

Thus, organodynamics has been constructed in order to fashion faithful models of a class of complex adaptive dynamical systems that other existing dynamical systems theory cannot model with a sufficient degree of fidelity – at least as far as I am concerned. The other existing theories do not unearth the fundamental ontology of the class of systems that organodynamics targets.

The Target Systems are Complex

Models of target systems must have the "right amount of" complexity. This is a kind of "Goldilocks principle" of modeling. If a model is too complex, then it fails to provide the intellectual accessibility that suits its usefulness. On the other hand, if it is too simple, then it fails to conserve certain salient features of the target system.

In other words, the complexity level if the model must be less than the target system that it represents, but it must be complex enough to exhibit the same salient features as the target system.

Unfortunately, the class of "target systems" that organodynamics targets contains exceedingly complex systems – biological systems being the chief exemplar. I have made the case that – to me - nonlinear dynamics is "too simple" to be able to conserve some of the principle salient features of biological systems – namely, their highly organized nature and their chance nature.

Of course, nonlinear dynamics is probably the right level of complexity for modeling the kind of systems that it targets – systems exhibiting "sensitivity to initial conditions". There is nothing wrong with nonlinear dynamics, or with organodynamics. The two theories simply target different classes of systems that exhibit different salient features.

So, if I am advancing a new theory that *is* sufficiently complex to be able to conserve those particular two salient features (organization and stochasticity), then it may well be that such a theory must be more complex than nonlinear dynamics.

Hopefully, organodynamics follows the prescription often attributed to Einstein, that the solution should be as simple as possible, but no simpler.

The best I can do is to try my hand at constructing such a theory, and hope that it somehow remains tractable – or at least that it can be approached by some tractable strategy.

Incremental Modeling

Of course, what we need is a strategy, or methodology, for "sneaking up" on the process of modeling complex systems using organodynamics. Such a methodology needs to be devised and formalized – and I regard such a project as a responsibility of mine. Nevertheless, for now there are a number of things that I can say about what such a modeling methodology should look like.

It should take an incremental approach. It should assume that a very simple model of a highly complex target system should start out as quite simple. But that a plan should be in place to gradually enhance the model – through a sequence of iterations, each of which adds a few features – until a model of the desired faithfulness is reached.

There are a number of standard iterative modeling techniques that can be employed throughout such an iterative approach that can be useful. I shall recount a couple of them by way of example in what follows.

It should be realized that statistical mechanics was plagued with every one of the challenges that I mention here for organodynamics. So organodynamics is not alone in this. Organodynamics can draw from statistical mechanics a number of techniques for incremental modeling of very complex spaces.

Course Graining

We noted earlier that the sample spaces of organodynamics are based on the set of all topologies of some underlying finite set of elements. This set of topologies of course can be a very large finite set. Moreover, probabilities will have to be assigned to each of these before the model can get off the ground.

Thus, it behooves the modeler to be able to define a reduced version of this set for early rendition of an incremental approach to modeling the target system.

One approach is *course graining*. This involves partitioning the set of all such sample points (topologies) into a set of categories of topologies. The early renditions of the model would use each category of topologies as a sample point, rather than the individual topologies. Very early rendition would use partitions that only have a few compartments. Each compartment would be understood as a "sample point" for that rendition. As later renditions progress, the re-partitioning of the set of all topologies into finer granularities would allow for greater numbers of compartments – each of which is a sample point. This approach would allow the number of sample points to start out as small, but increase over time. Thus, the granularity of the model over time would change from course-grained to fine-grained.

Theoretical Probability Assignments

Functionally, it would seem that one must empirically observe the target system in action and record relative frequencies of the sample point realizations in order to estimate the probabilities of each, and thus the probability distribution for the whole sample space. Such a task is clearly impossible for the class of complex systems targeted by organodynamics.

A realistic alternative is to assess the particular nature of the target dynamical system at hand, and then to identify some theoretical probability distribution that has been defined in a way so as to exhibit that nature.

Statistical mechanics, for example, had exactly this problem and approached it in exactly this manner. In other words, where empirical observation was intractable for statistical mechanics, a theoretical approach to probability assignment was a reasonable alternative.

Boltzmann, for example, assumed an "equilibrium condition" in which the probabilities of every state were equally likely. J. W. Gibbs later defined other sets of conditions, which implied other distributions. These assumed conditions he called "ensembles" [Tolman 1938].

Theoretical Probability Refinements

Once a theoretical distribution has been selected for an initial rendition of an organodynamics model, its veracity can be subjected to empirical tests. Such tests typically compare the theoretical probabilities to a few observed probabilities.

After the conduct of such test, the probabilities provided by the theoretical distribution are revised. This revised distribution is then used in a second incremental rendition – as though it were a new theoretical distribution, and again compared to observed probabilities. This iterative process continues incrementally, each rendition getting increasingly complex and increasingly faithful.

A number of formalizations of this general approach have been attempted. [Jaynes 1957] advocates the application of Bayesian analysis to this approach. [Majda, et. al. 2014] uses an approach based on information theory generally, and relative entropy specifically.

Summary of Organodynamics

Organodynamics is a complex adaptive dynamical systems theory that seeks to provide an alternative for modeling a certain class of dynamical systems. The inspiring exemplar of this class is biological systems.

Organodynamics as a Dynamical Systems Theory

There are three salient feature of this target class that organodynamics seeks to model:

1. System organization
2. Change of system organization (reorganization)
3. The chance nature of reorganization.

As a systems theory, organodynamics defines *system state* as *organization*; and *trajectory* as *reorganization*. Trajectory in organodynamics is a chance variable.

State in organodynamics is defined as system organization. Organization is articulated as an *extended topology*. The state space is a set of extended topologies – one for each possible way that the system can be organized.

Chance is introduced into organodynamics by assigning a probability to each of these states. Thus, the state space becomes a probability space. When extended over time, the probability space becomes a discrete stochastic process.

Organodynamics and Information Theory

Since the sample space of organodynamics is a “set of topologies”, then there is no natural, or semantically useful, mapping of the sample space to the real numbers. This means that the concept of *mean* or *expected value* is not mathematically defined for organodynamics. This is because the definitions of both *mean* and *expected value* require a real number to be associated with each sample point. For the same reason, none of variance, standard deviation, or any moments or central moments is defined either.

Of course, these statistics are collectively a member of the mechanisms of mathematical statistics (mean, variance, etc.) known as *moments* and *central moments*, which constitute a central mechanism of mathematical statistics for characterizing chance variation. But, as we have just discussed, these statistics (central moments) cannot be defined for organodynamics, since the sample points of organodynamics are not real numbers and cannot be mapped to real numbers in any semantically useful way.

This means that the central mechanism of mathematical statistics does not provide useful tools to characterize chance fluctuations in organodynamics. Thus, organodynamics must find another branch of probability theory (than mathematical

statistics) that is also capable of characterizing chance variation – using some other tools than moments and central moments.

Fortunately, information theory is such a discipline.

Information theory is a branch of probability theory that characterizes probability spaces using a set of functionals called *entropic functionals* [Cover and Thomas 1991]. The only inputs required by entropic functionals are *probabilities*. No real valued mappings of the sample points (i.e. random variables) are required.

Information Theory provides the mathematical foundation of organodynamics.

Information theory defines a number of entropic functionals to characterize chance fluctuation (variation) in a probability space [Cover and Thomas 1991. These functionals measure the degree of uncertainty inherent in a probability distribution (space) [Kleeman 2009]. These functionals are named: entropy, joint entropy, conditional entropy, relative entropy, mutual information and entropy rate.

Like the moments and central moments of mathematical statistics, these entropic functionals characterize the degree of chance variation in a probability space (distribution) - but use only probabilities as inputs. They do not require a real-valued function, or random variable (in the strict sense of the term).

The key to constrained or controlled dynamics in random processes is stochastic dependence. Information theory provides strong support for characterizing the constrained behavior of stochastic processes. This support especially includes: *conditional entropy*, *relative entropy* and *entropy rate*.

In organodynamics, a dependent stochastic process that is called an organodynamic dependent stochastic process, or ODSP describes the “dynamics” of a target application. An ODSP is articulated as a sequence of transition matrices, one for each times step in the ODSP. In typical cases, the Markov condition can be assumed, and the ODSP becomes an organodynamic Markov chain, or OMC.

An organodynamic process is often initially modeled as a *piecewise homogeneous* ODSP. However, such a process can be “homogenized” into a homogeneous OMC.

Combining Organizational Dynamics and Information Theory

Organodynamics combines organizational dynamics with entropic functionals to model biological and other highly complex dynamical processes perhaps more faithfully with respect to organizational state and stochastic dynamics than nonlinear dynamics.

Organodynamics does this by defining: OSS, OPS, OPD, ODPD, OSP and ODSP - and by characterizing long-term behavior in the ODSP. Often, and ODSP can be reduced to the more tractable OMC, or *organodynamic Markov chain*.

An ODSP is a dependent stochastic process - e.g. a Markov process - whose sample points are extended topologies as defined by organodynamics. One of these ODSPs defines the dynamics of an organodynamics application.

Defining the dynamics of the application amounts to defining the transition matrices of the ODSP.

Complexity Modeled Faithfully through Probability Alone

Organodynamics seeks to be a complex adaptive dynamical systems theory that describes the dynamics of a certain class of highly complex systems by probability alone.

This modeling capability intends to leverage probability theory alone to describe the conditions under which the time evolution of systems can be constrained sufficiently well so as to permit prediction.

However, certain complex behaviors, such as adaptation, promote persistence of the process, while at the same time resisting predictability. These are extremely complex systems whose behavior is constrained, while at the same time are non-predictable in nature. They cannot be predictable because they are able to respond to unpredictable events in the environment in such a constrained manner that they promote their own persistence – even while being completely describable by probability constructs alone.

I propose that evolutionary biology is an existence proof of this assertion.

Organodynamic has been constructed as a dynamical systems theory so as to model these kinds of phenomena.

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