# Intelligent Evolution, Complexity and Self-Organization

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**ABSTRACT**

Have physicists found the underlying science driving the origin and evolution of life? It follows from the fundamental laws of nature. From this standpoint, there is one essential difference between living subjects and inanimate carbon atoms: The former tend to be much better at *capturing energy from their environment* and *dissipating* that energy as heat. The mathematical formula, based on established physics, indicates that when a group of atoms is driven by an external source of energy and surrounded by a heat bath, will often gradually *restructure itself* in order to dissipate increasingly more energy. Under similar conditions, matter inexorably acquires the key physical attribute associated with *life.* From the perspective of the Prigogine-England physics, Darwinian evolution is only a special case of more general phenomenon, including also the controversies in oncology. The ongoing Intelligent Evolution (IE) will develop the full Artificial Intelligence (AI).

**Key Words:** evolution, life, Prigogine-England physics, artificial intelligence, dissipation, energy
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## Introduction

Theoretical results of physicists from Prigogine to England are generally considered valid. Their formula represents *the driving force behind* a class of phenomena in nature that includes life. A system of particles is driven by an oscillating force. Over time (top-down), the force triggers the formation of more bonds among the particles. At the heart of Prigogine-England theory is the second law of thermodynamics, the law of increasing entropy -the arrow of time- (Prigogine, 1997). The energy tends to disperse or spread out as time progresses. Entropy is the measure of this tendency. It increases as a matter of probability: There are more ways for energy to be spread out than for it to be concentrated. The entropy must increase over time in an isolated or "closed" system-an "open" system can keep *its* entropy low-that is, divide energy unevenly among atoms-by greatly increasing the entropy of its *surroundings.* In his influential 1944 monograph "*What Is Life?*" physicist Schrödinger argued that this is what living systems must do. The overall entropy of the universe increases during photosynthesis as the sunlight dissipates, even as the plant prevents itself from decaying by maintaining an orderly internal structure. In the late 1990s, Ch. Jarzynski and G. Crooks showed that entropy produced by a thermodynamic process corresponds to a simple *ratio*: the probability that the atoms will undergo that process divided by their probability of undergoing the reverse process. As entropy production increases, so does this ratio: A system's behavior becomes more and more "irreversible". Particles tend to dissipate more energy when they *resonate with a driving force.*

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"This means clumps of atoms surrounded by a bath at some temperature, like the atmosphere or the ocean, should tend over time to arrange themselves *to resonate better and better* with the sources of mechanical, electromagnetic or chemical *work in their environments.*" (England, 2013).

## Complexity and Self-Organization

*Self-replication* (or reproduction-in biological terms), the process that drives the evolution of life, is one such mechanism by which *a system might dissipate* an increasing amount of energy over time. The theoretical minimum amount of dissipation that can occur during the self-replication of RNA molecules and bacterial cells is very close to the actual amounts these systems dissipate when replicating.

According to Prigogine-England's theory, the underlying principle driving whole process is *dissipation-driven adaptation* of matter. This principle would apply to *inanimate matter as well.* Besides *self-replication, greater structural organization* is another means by which *strongly driven systems* ramp up their ability to dissipate energy. England argues that under certain conditions *matter will spontaneously self-organize.* The distinction between living and nonliving matter is not sharp. This new facts will have impact on *ecology*, *robotics* and *environment* at all, including *cancer microenvironment* too.

England's theory could be tested by comparing cells with different *mutations* and looking for a correlation between *the amount of energy* the cells dissipate and their *replication rates.* Connection between England's theory with Brenner's *microsphere constructions* may show whether the theory correctly predicts which self-replication and *self-assembly* processes can occur, is probably *a fundamental question in science* today.

Conventional wisdom holds that *complex structures* evolve from simpler ones, by *step-by-step method,* through a gradual evolutionary process, with *Darwinian selection* favoring *intermediate forms* along the way. But recently a growing number of scholars have proposed that *complexity* can arise also *by other means*-as a *side effect,* for example-*even without natural selection* to promote it. Studies suggest that *random mutations* that individually have no effect on an organism can *fuel the emergence of complexity* in a process named as *constructive neutral evolution.*

Complexity can arise through other routes, because *life has a built-in tendency to become more complex* over time. It has been newly shown that *random mutations arise; complexity emerges* as a *side effect, even without natural selection.* For example, McShea and Brandon in their book *Biology's First Law* argued that bunch of parts that start out more or less the same should *differentiate* over time. When organisms *reproduce,* one or more of their *genes* may *mutate.* These mutations give rise to more *type of the parts.* If an organism has more parts, those units have an opportunity *to become different.* After a gene is accidentally *copied, the duplicate may pick up mutations* that the original does not share. If you start with a set of identical parts, they will *tend to become increasingly different* from one another. It means, *the organism's complexity will increase.*

As *complexity arises,* it may help an organism *survive better* or *have more offspring.* If so, it *will be favored* to develop and *spread* through the population. The new copies will mutate. Natural selection will keep these mutations from spreading through populations. That is, organisms born with those traits *will tend to die* before reproducing, thus taking these traits *out of circulation.* So far, *natural selection works against complexity.*

Unlike standard evolutionary theory, we can see *complexity increasing* even in *the absence of natural selection.* It maintain a fundamental law of biology-perhaps its only one-*the zero-force evolutionary law* (McShea and Brandon, 2010).

Consequently, natural selection is strong among the organisms living in wilder, *eliminating mutations* that make, for example, flies unable to cope with their many challenges. In *sheltered environment* of the labs, in contrast, *natural selection is feeble.* Lab-raised fruit flies are *more complex* than wild ones because their *sheltered environment allows even disadvantageous mutations to spread.* The zero-force evolutionary law makes a clear prediction: over their multiple use in research, the lab flies should have been *less subject to the elimination of redundant (disadvantageous) mutations* and thus should have *become more complex* than the wild ones.

An organism can *exist without external selection*-without the environment determining who *wins and losses in the evolutionary race*-but it can be *subject to internal selection,* which takes place within organisms. *Freed* from natural

selection, organisms have *reveled in complexity.* For this type of evolution is an exact label *"constructive-neutral evolution."* Experiments may decide why we don't just have genes with the correct original sequence, making *RNA editing* unnecessary, etc. Further research will coax multiple *biological theories* to *think beyond natural selection* and to see the possibility that *random mutation* can fuel *the evolution complexity* on its own. Our point is that we *don't dismiss adaptation;* we just *don't think it explains everything.*

## Free Energy and Darwinian Selection

The free-energy construct was introduced into statistical physics to *convert* difficult *probability-density integration* problems into easier *optimization* problems. In the present context, free energy provides the answer to a fundamental question: how do *self-organizing adaptive systems* avoid surprising states? (Friston, 2010). Our answer is: they can do this by *optimizing* (not only minimizing, suppressing, i.e., Darwinian selection) their free energy.

Complexity is the difference between *the recognition density* and *the prior density* on causes. The difference between the prior densities (encodes beliefs about the state of the world before sensory data that are *assimilated*) and the recognition density (encodes posterior beliefs) (Friston, 2010). The agent will *selectively sample* the sensory inputs that it *expects:* we *anticipate* what might be next and then try to *confirm those expectations.* So, when we are *adapting to the environment,* we *conform to expectations.*

In *hierarchical models* causes in one level *generate subordinate causes* in lower level (sensory data *per se* are generated at the lowest level). Using the free energy effectively *optimizes* empirical priors, i.e. the probability of causes at one level is given those in *the level above.* Because empirical priors are *linked hierarchically,* they are informed by sensory data, enabling *the brain to optimize* its prior expectations *online.*

The units in the level *above* encode conditional expectations which optimize *top-down predictions* to *explain away* (to reduce) prediction error in the level *below.* It means *excitatory bottom-up* inputs to a prediction error neuron with *inhibitory* synaptic inputs that are *driven by top-down predictions.* In this *hierarchical arrangement (adaptive resonance theory)* the problem arises because they *ignore uncertainty and decoherence* in *probabilistic representations.* This is because the parameters of hierarchical model *determine* how expected states (synaptic activity) are *mixed* to form predictions *trade-off.* So far, the prediction errors in level above can by *top-down links suppress* the predictions in level below. This *centralized elimination, repression, suppression, resonance, assimilation; dissipation* is *the core of Darwinian adaptation* is a *hierarchical conformism.* That is why may be Darwinian adaptation only a *special case* of *the broader theory of adaptation.*

Another problem with the Darwinian adaptation is given by *Quantum Darwinism* and phenomenon of decoherence. It means that any *coherent superposition* of the system's quantum states is continuously *reduced to a mixture.* A preferred basis called a *"pointer basis"* is singled out. An effective *counterselection rule* has emerged: the decoherence which prevents superpositions of the preferred basis from persisting by a *lost of diagonal terms of the density matrix.* Reduction to a mixture can't be interpreted as adaptation, progress, development, forward evolution of the system. It is clearly only a counterselection.

In this case the density matrix *p(x,x')*of the particle in the position representations *evolves* according to the master equation

$$\frac{dp}{dt} = -\frac{i}{\hbar}[H, p] - \gamma(x - x')\left(\frac{\partial p}{\partial x} - \frac{\partial p}{\partial x'}\right)$$
$$-\frac{2m\gamma k_B T}{\hbar^2}(x - x')^2 p$$

where $H$ is the particle's Hamiltonian, $V(x)$ is potential, $\gamma$ is the relaxation rate, $k_B$ is the Boltzmann constant, and $T$ is the temperature of the field. This equation naturally separates into three distinct terms, each of them responsible for a different aspect of *classical behavior*: the first term, the von Neumann equation is derived from the Schrödinger equation. The second term causes *dissipation:* the loss of energy and *decrease of the average momentum.* The third term is responsible for the fluctuations or *random* kicks that lead to Brownian motion (Zurek, 1991). They causing *the environment-induced decoherence based counterselection.*

*Negative selection* due to decoherence is the essence of *environment-induced*

*counterselection of Darwinian adaptation.* Under scrutiny of the environment, only pointer states remain unchanged. Other states decohere into mixtures of stable pointer states that can *persist,* and, in this sense *exist:* they are *counterselected.*

Only states that produce *multiple informational offspring – multiple imprints on the environment* – can be found out from the fragments of environment. The origin of the emergent classicality is then *not just survival of the fittest* states to *deposit multiple records* – copies of themselves – through environment. *Proliferation of records* allows *information about system* to be *extracted* from many fragments of the environment. Thus, environment acquires *redundant records of the system* (Zurek, 2009).

## The Inhibitory-Proliferative Adaptation

In our model the values of $y_1$ and $y_2$ are transformed through a nonlinear activation function $f(y)$ before they inhibit each other:

$$dy_i = \left[ -ky_i - w\sum_{j=I}^{N} f(y_j) + I_i \right] dt + c_i dW_i,$$

integration starts from $y_i(0) = 0$, an input unit with mean activity $I_i$, and independent white noise fluctuations $dW_i$ of amplitude $c_i$. These units also inhibit each other with a connection weight $w$, $k$ denotes the decay rate of the accumulated activity with leak (entropy), $N$ means the number of alternatives. The inhibition parameter $w$ *suppress* the self-replicators $y_3$, $y_4$, $y_5$ - i.e., *counterselect* the $y_1$, $y_2$.

From a physiological perspective, increased *methylation* of the *Avp* enhancer during postnatal life serves to *restrain the HPA axis* in critical periods when *homeostatic thresholds* are set, what *facilitate adaptation* of the endocrine system to environmental stimuli. Phosphorylation of MeCP2 is a conduit of *experience-driven changes* in *gene expression,* serving as an important mediator. Focusing on *DNA methylation,* provide evidence for post mitotic epigenetic modifications in *neuronal function,* which facilitate *physiological* and *behavioral adaptations* (Murgatroyd *et al.,* 2009).

## Conclusions

Despite of Darwin, England and Hawking, for the ongoing type of evolution we propose the label *"inhibitory-proliferative adaptation"* as a form of *Intelligent Evolution (IE)* leading to the development of full *Artificial Intelligence (AI).* "The generation of nonequilibrium structures (such as Bénard vortices or chemical oscillations), where energy is conserved, also corresponds to a free lunch, *for the price of nonequilibrium structures is entropy,* and not energy." (Prigogine, 1997).

*The hierarchical arrangement of adaptation* with *brain at the top* may be in *contradiction* with recent research focus on *tumor microenvironment* leading to *controversies in oncology.*

The brain plays critical role in appraising *social stressors,* as well as in *modulating the immune system's response to stressors* that involve *social* or *physical threat.* Differences of *inflammatory responses* to social stress depend on individual differences in activity of *neural regions* that process *social threat-related information.* Stressors of *social rejection up-regulate* inflammatory activity. The neural regions involved in processing *rejection-related distress* relate to individuals *magnitude* of inflammatory responses to social stress.

General *stress-response-controlled switch* to *error-prone DNA break* (double-strand break, DSB) *repair-dependent* stress-induced mutation, driven by spontaneous DNA breaks, is pathway that cells use and a major source of *spontaneous mutation.* Mechanisms that couple *mutagenesis to stress responses* allow cells to *evolve rapidly* and *responsively to their environment.* Furthermore, stress is not required; *activation of the stress response is sufficient* (Shee *et al.,* 2011).

On the basis of prior research, these brain regions include *the dorsal anterior cingulated cortex* (dACC), and *the anterior insula.* Exposure to an acute episode of *social rejection* or to rejection-related cues (for example the unemployment) has been shown to activate both the dACC and the anterior insula. Greater activity found in the dACC, in turn, has been associated with greater *self-reported feelings* of *social distress* (Slavich *et al.,* 2010).

*Psychological stress* and exposure to the stressor of Darwinian *social disruption (SDR),* as *a cause of carcinogenesis,* increase cytokine

production by monocytes/macrophages and reduce their sensitivity to corticosterone. Repeated *social defeat* during SDR resulted in a significant increase in spleen mass and the number of splenic monocytes/macrophages and granulocytes. It indicates that repeated *social defeat* during the SDR stressor enhances innate immunity to *E. coli* infection and SDR significantly impacts splenic monocytes/macrophages.

In settings in which *hierarchies are strongly enforced,* and *subordinates* have little social support, *low dominance rank* can lead to *chronic stress, immune compromise,* reproductive *dysregulation* and *cancer* (Murgatroyd *et al.,* 2009; Shee *et al.,* 2011; Tung *et al.,* 2012).

It is widely accepted that *psychological stress* affects *the immune response,* and *chronic, repeated* exposure to a stressor is *immunosuppressive.* Ligation of glucocorticoid (GC) receptors on mononuclear cells *suppresses* the expression of cytokines, chemokines, and adhesion molecules through negative regulation of NF-$\kappa$B activation and function (Bailey *et al.,* 2007).

Recent experimental studies have illuminated *the mechanistic pathways* by which NF-$\kappa$B signaling contributes to the aspects of *carcinogenesis.* These data showing that *chronic inflammation promotes carcinogenesis* and that NF-$\kappa$B signaling is at the heart of such inflammation. Despite versatile and occasionally *antagonistic interactions,* NF-$\kappa$B and STAT3 *cooperate to promote* the development and progression of colon, gastric and liver *cancers.* IE means a *transition* from *the hierarchical arrangement* to *the parallel networking.* The hierarchical arrangement leads to stressor *social disruption* which is subsequently *contributing to the carcinogenesis.*

## References

Avitsur R, Stark JL, Dhabhar FS, Sheridan JF. Social stress alters splenocyte phenotype and function. J Neuroimmunol 2002; 132: 66-71.

Bailey MT, Engler H, Powell ND *et al.* Repeated social defeat increases the bactericidal activity of splenic macrophages through a Toll-like receptor-dependent pathway. Am J Physiol Integr Comp Physiol 2007; 293: R1180-R1190.

England JL. Statistical physics of self-replication. J Chem Phys 2013; 139: 121923/1-8.

Engler H, Engler A, Bailey MT, Sheridan JF. Tissue-specific alterations in the glucocorticoid sensitivity of immune cells following repeated social defeat in mice. J Neuroimmunol 2005; 163:110-119.

Friston K. The free-energy principle: a unified brain theory? Nature Reviews Neuroscience 2010; doi: 10.1038/nrn2787:1-12.

Mays JW, Bailey MT, Sheridan JF *et al.* Influenza Virus-Specific Immunological Memory Is Enhanced by Repeated Social Defeat. J Immunol 2010; 184 (4): 2014-2025.

McShea DV, Brandon RN. Biology's First Law. The University of Chicago Press, 2010; p.184.

Murgatroyd C, Patchev AV, Wu Y *et al.* Dynamic DNA methylation programs persistent adverse effects of early-life stress. Nature Neuroscience 2009; doi:10.1038/nn.2436: 1-10

Prigogine I. The End of Certainty. Time, Chaos, and New Laws of Nature. The Free Press, 1997; p.175.

Shee C, Gibson JL, Darrow MC *et al.* Impact of a stress-inducible switch to mutagenic repair of DNA breaks on mutation in Escherichia coli. PNAS 2011; doi: 10.1073/pnas.114681108: 13659-13664.

Slavich GM. *et al.* Neural sensitivity to social rejection is associated with inflammatory responses to social stress. PNAS 2010; doi/10.1073/pnas.1009164107: 1-6.

Stark JL, Avitsur R, Sheridan JF *et al.* Interleukin-6 and the development of social disruption-induced glucocorticoid resistance. J Neuroimmunol 2002; 124: 9-15.

Tung J, Barreiro LB, Johnson ZP *et al.* Social environment is associated with gene regulatory variation in the rhesus macaque immune system. PNAS 2012; doi/10.1073/pnas.1202734109: 109(17): 6490-6495.

Zurek WH. Decoherence and the Transition from Quantum to Classical. Physics Today 1991; 36-44.

Zurek WH. Quantum Darwinism. arXiv:0903.5082v 1 [quant-ph] 29 Mar. 2009; 1-10.