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Commentary

How do biological systems escape 'chaotic' state?

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Chaos theory specifies that processes with small change in a state of the deterministic nonlinear system result in large variations in the final states (Boeing 2015; Kellert 1993). That means small changes in the initial conditions can give rise to large differences in the final states of a system resulting into the so-called 'chaotic' state! Chaotic behavior is highly widespread in the universe such as in weather and climate (Lorenz 1963; Ivancevic and Ivancevic 2008), arising spontaneously in some artificial states such as road traffic, etc. (Safonov *et al.* 2002). So much so that chaos theory has found applications in disparate disciplines of science such as meteorology, anthropology (Mosko and Damon 2005; Trnka and Lorencova 2016), sociology, physics, computer science, economics and even biology (Hubler 1989). The theory spawned such fields of study as complex dynamical systems, edge of chaos theory, and self-assembly processes, etc.

Are biological systems chaotic and whether biological processes that are intrinsically 'noisy' and sensitive to external conditions are extensively prone to chaotic effects? Clearly, this is not the case to a large extent: Biological systems exhibit fair degree of robustness in regulating themselves, cell-autonomously as well as cell non-autonomously and rarely 'tip into' chaotic states in normal biotic or physiological contexts. How do they manage this 'fete' in spite of extremely high nonlinear dynamic complexity associated with them at multiple levels? Is there a system-level property that can explain this quandary across multiple scales of biological system?

Biological designs make sense only in the context of Evolution and Ecology that they are part of. Organisms and cells can exhibit multiple phenotypes that are plastic as well as robust at the same time even within a single genotype, portending that the systems are almost on the verge of chaos, but not quite tipping over to the state of 'chaos' in normal ambient conditions. Do we understand this? Biological homeostasis seems to be a reasonable explanation that resolves this quandary.

Homeostasis is a physiological response that confers stability to the system by the cumulative action of dynamic feed forward and feedback regulations among several interdependent components of the system, such that the system stays 'quasi-stable' at the expense of constant energy inputs. Homeostasis is revealed in a chair-shaped graphical relationship between environment or genotype (independent variable) and the resultant phenotypes (dependent variable). The homeostatic plateau (in the dependent variable) is the region where active mechanisms intervene and stabilize the phenotype (Nijhout *et al.* 2017). It is surmised that mutations destabilize homeostasis and reduce the size of the stable range (homeostatic plateau), thereby triggering 'escape from homeostasis' (Nijhout *et al.* 2014) where phenotypes begin to become less stable and eventually turn into fully unstable state, the start of 'chaos' in a system. Therefore, the key to biological designs is to stay close to or within the 'homeostatic plateau' and resist drifting into 'chaos'. How is it achieved?

Since many different phenotypes can correspond to a single genotype, one would wonder how selection could operate through the genome impacting multiple phenotypes. It is proposed that the homeostatic mechanisms operate within a limited range such that outside the limited range, the controlled variable changes rapidly allowing natural selection to act. Mutations and environmental stressors can disrupt homeostatic mechanisms, exposing cryptic genetic variation upon which natural selection can act. Therefore, it is plausible that homeostatic mechanisms buffer traits against environmental and genetic variation and thereby allow accumulation of cryptic genetic variation. There are homeostatic regions (the plateau region discussed above) where the trait is relatively insensitive to genetic or environmental variation (i.e. stable phenotype), flanked by regions where it is sensitive (regions where the plateau deviates) (i.e. less stable or unstable phenotypes). Phenotypes that fall far away from 'plateau' come under natural selection and eventually get eliminated (or selected in varying external conditions), thereby unveiling a 'Darwinian filter' that tends to keep Biology close to homeostasis plateau. Extensive biochemical, genetic and cellular data have now uncovered complex regulatory circuits that interconnect several biological pathways (processes) underscoring robust feedback/feed-forward regulatory circuits mediating the homeostatic control mechanisms, maintaining the 'homeostatic plateaus'.

Keywords. Chaos; Darwinian; homeostasis; phenotypes; regulation

Motivated by the data as well as by the theory, recently developed models suggest that control and synchronization can also be achieved even in chaotic dynamically complex networks when feedback regulations become ‘adaptive’ as well as ‘deterministic’ (Lin *et al.* 2017) as in biological systems. In a highly coupled interactive system (cluster), if stochastic forces can manage to affect individual components selectively, the noise associated with such stochastic forces can in fact enhance the cluster size (Clusella and Politi 2017), which then adds to the fitness strength of the cluster (biological system).

All such unintuitive results demonstrate that in Biological world, evolution, in the context of prevailing ecology, fine-tunes mechanisms that greatly stabilize ‘homeostatic controls’. The power of homeostatic control mechanisms are all pervasive in biology: Signaling associated with kinase-phosphatase circuitry, activation-inactivation marks on chromatin, anterograde-retrograde signaling in cells, inter cellular & inter tissue communications via soluble factor signaling in animals, photosystem adaptations in plant chloroplasts during high-light to low-light (or vice versa) transitions, cross-tuning between carbon concentrating mechanisms (CCM) and photorespiration (PR) in plant cells and inter-organismal cross-talk emanating in predator-prey dynamics at population level interactions, are all rich examples of the balancing acts that lead to Homeostatic controls in Biology.

It is very likely that various tissues in an organism operate at differing degrees of homeostasis fidelity controls whose mechanistic basis needs to be better appreciated. It is relevant to point out that cardiac rhythmicity controls (Sharma 2009) as well as neuronal activity modulations (Paul *et al.* 2016) are well known, but rare, examples of tissue responses that exhibit discernible level of ‘chaotic’ patterns in certain contexts for generating physiologically relevant functional outputs. In the context of aging, one would surmise that the organism as a whole and/or various tissues of the organism differentially get subjected to gradual decline in the fidelity of homeostatic controls, whose molecular basis may uncover interesting new insights. Finally, genome landscapes of all organisms are constantly under the ‘grip of chaos’ due to ongoing ‘genetic drifts’ (Lynch and Cooney 2003) during evolutionary time-scales!

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