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MEMORY EVOLUTIVE SYSTEMS: AN APPLICATION TO AN AGING THEORY

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Memory Evolutive Systems have been introduced by the authors as a mathematical model (based on Category theory) for natural open self-organizing systems such as socio-biological or neural systems. The architecture of a MES is a compromise between a parallel-distributed processing with a modular organization, and a hierarchical associative network. The complex dynamics is modulated by the competitive interactions between a family of internal Centers of Regulation (CR) each with its own complexity level and time-scale. Their 'dialectics' through functional loops is responsible for the development of higher order cognition (EV91, 92). Here it is exploited to elaborate a model of 'aging by spreading desynchronisation' for complex systems: Sequences of local changes to counteract stochastic external perturbations increase the turnovers and the propagation delays; whence higher and higher CRs are forced to lengthen their periods in order to maintain their deterministic structural 'temporal constraints'. This theory unifies most of the known theories of aging for organisms.

1. A brief Review of Memory Evolutive Systems.

Let us recall the definition of a MES (cf. EV87, 90, 92). The state of the system at a given time is modelled by a category, formed by its components and their interactions (transfers of informations, energy or constraints). The system has an organizational hierarchy, with its objects separated into various complexity levels: an object of level k+1 is the cohesive binding (or 'inductive limit' in the category, Kan 1958; cf. also EV87) of the pattern representing its internal organization, i.e., formed by its own components of level k and their specific links.

The changes of state are represented by functors between successive state-categories. The dynamics is regulated by a family (CR_n) of sub-systems, the Centers of Regulation, operating in parallel, but each with its own complexity level, time-scale, and differential access to a central hierarchical sub-system, the Memory. The higher centers supervise more specialized lower centers.

The trial-and-error learning for each CR_n processes stepwise. At each step, CR_n as an observational organ, constructs a category P modelling its own representation of the global system, called its *actual landscape*. As a command organ, its components (called actors) coordinate their 'goals' to select a strategy Σ on P consisting in the addition or subtraction of some elements, disassociation of complex objects, cohesive binding of some patterns (e.g., by strengthening of their links) so that they become new (complex) units of a higher level. The anticipated landscape P' at the end of the step should be the 'complexification' of P with respect to this strategy (constructed in the categorical setting, cf. EV87). However, the strategies of the different centers being competitive, Σ might not

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be implemented so that there would be a difference between P' and the 'real' landscape at the regular end of the step, or earlier if a 'fracture' forces the center to interrupt the step. CR_n, as a control organ, measures this difference (by the comparison functor), and memorizes it at the next step.

2. Temporal constraints imposed by the functional loops between levels.

The specific time-scale of each CR_n determines the duration of the successive steps of its learning process. We denote by $d_n(t)$ the *period* of CR_n at the date t, which is the mean length of the steps preceding t. This period increases with the complexity level of the center. It must satisfy temporal constraints (given below) which ensure that: 1. the actors have enough time to form the actual landscape, communicate to choose a common strategy, send commands to the effectors to enact it, control its results and eventually adjust it anew; and 2. the components in the landscape preserve their overall internal organization up to the end of the step, in spite of the turnover of their lower order components.

To each link in the system is associated a real number, representing its *propagation delay*, which increases with the complexity level of the components it links. We denote by $p_n(t)$ the mean propagation delay of the links which intervene in the actual landscape of CR_n at the time t.

The *stability span* at t of a component B of level k is defined as the greatest number δt such that there is a pattern of level k—1 having B as its cohesive binding at t and whose image by the change-of-state functor admits the new state of B at $t+\delta t$ as its cohesive binding (EV 87, page 37). It is inversely related to the rate of change at the lower level $k-1$, and its order of magnitude increases with k. For a population of proteins, it would be proportional to its half-life. We denote by $\tau_n(t)$ the mean of the stability spans of the components intervening in the actual landscape of CR_n at t.

For the steps to be completed in time, the following *structural temporal constraints* must be satisfied: *For each center* CR_n *and for almost all* t *(i.e. except on a set of measure* 0*), the order of magnitude of the period* $d_n(t)$ *must be greater than that of* $p_n(t)$ *and lesser than that of* $\tau_n(t)$, that is:
$\tau_n(t) >> d_n(t) >> p_n(t)$ (>> means 'is of an order of magnitude greater than').

There is a *desynchronisation* of CR_n if its period must be lengthened in order to maintain these constraints after p_n or τ_n have been modified.

3. A model of Aging by spreading Desynchronisation.

Aging for a MES will result from the interplay between stochastic external events and the deterministic structural temporal constraints of its CRs.

For an organism, we consider the MES of its components of all levels: sub-molecular, macromolecular, infra-cellular, cellular, tissues, organs and large systems (immunological, circulatory, nervous,...), each with its own time scale. External perturbations (UV radiations, dietetical factors...) cause fractures for some lower center. If its own built-in repair mechanisms are overrun, a recourse is attempted to higher level repair processes (for instance the SOS system for DNA repair, Radman 1975). But this lengthens the propagation delays and implies sequences of lower level changes which increase the turnovers and so decrease

the stability spans of higher components, with a risk that a higher center be impeded to react soon enough. To maintain its structural temporal constraint (whose prolonged default means death), this center must lengthen its period. The process spreads to higher centers, leading to a cascade of desynchronisations.

This scenario is in agreement with most of the physiological theories of aging (cf. Robert 1983, and H1990), which differ by the level of attack: submolecular (free radical theory), macromolecular (Orgel (1963) cascade of catastrophe), infracellular or cellular (e.g. Szilard stochastic mutations or Hayflick deterministic number of replications), tissues (degradation of the extracellular matrix).

It suggests the following model of *Aging by spreading desynchronisation*, also valid for a social group, an ecosystem, a company, ... In a MES, *aging is characterized by the fact that, for some* CR_n *the ratios* $\tau_n(t)/p_n(t)$ *and* $\tau_n(t)/d_n(t)$ *decrease for almost all* t, *forcing successive desynchronisations on the CRs to maintain their structural temporal constraints*. It is pathological if these cannot be restaured soon enough at least at one level. This definition encompasses both the case where the propagation delays p_n increase, or the stability spans τ_n decrease (aging mechanism proposed in EV87; cf. also Rosen 1978), and in fact the variation of one factor repercutes on the second.

Analytically, when smoothed, d_n may be interpreted as the period of an oscillatory process for a limit cycle in a dissipative system with multiple regulations (Prigogine-Stengers 1979); the changes in τ_n and p_n correspond to a modification of the initial conditions, that might lead to a brief chaotic state, before reverting to an oscillatory process for another limit cycle whose longer period is that of CR_n after desynchronisation (equations are similar to Goldbeter 1990).

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