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## EPILOGUE

Electric bipolarity originated from the separation of the first opposite electric charges of negative electrons and positive positrons from neutral energetic photons. However, such symmetry was then broken and this original electric charge parity violated with the “overpowering” of matter over antimatter. Electric symmetry of matter was recovered with the advent of another positive partner for the electron resulting from the confinement of quarks into a unit of positive electric charge, namely the proton. The symmetry of charge thus recovered was however paralleled by an asymmetry of mass, the proton being 1840 x heavier than its negative counterpart, the electron. It is this event which can be considered as crucial as it gave birth to the primordial atom, hydrogen (H), and thereby the prototype of the electric atomic and then molecular dipoles by further complexification from the biogenic H<sub>2</sub>O toward the most sophisticated biopolarized structures spanning the evolutionary ladder.

All primordial polarities can be considered as passive or *intrinsic* to matter, from the intra-atomic mass disparity between the electron and the proton to the inter-atomic organization of the molecules as well as macromolecules. In contrast, active or *acquired* biopolarities are the result of polarization processes such as they intervene by energetically-driven charge separation in the cellular membranes which then exhibit an electric bipolarity. Similarly, they are also active polarization processes those which break the symmetrical content of initially homogeneous cells and thereby lead them to the classical example of bipolarly axiated spores and eggs of plants and animals. In short, passive polarities and active polarizations have in common the mediation of a symmetry-breaking process but differ in the fact that this asymmetry is established once for all in the first case while it must be re-created each time in the second case for every new polarization. It is at this level that symmetry-breaking processes associated with the dissipative structures described by Prigogine's school might be implicated. Such symmetry-breaking instabilities arising by a bifurcation mechanism are a most important property of those dissipative structures arising in far-from equilibrium conditions and are thus implicated in the onset of polarity.

We can then address the important question: can we extrapolate from *electric* to *biostructural* polarities involving gradiental distribution of morphogens? These questions can be tentatively answered by the two extreme points of view expressed by developmental biologists and summarized by Meinhardt (1982, in **I**):

1) Driesch's (1899) assumed that the overall orientation of the dorso-ventral (D-V) axis of a sea urchin egg results from the alignment of individual polar elements arranged like the dipoles of a magnet (see same comments about plants by Sinnott (1960 in **I**). Similarly, Harrison (1921) attributed the origin of overall polarity to the superposition of many small polar, proteinaceous structures. Meinhardt also considered (p. 37) as a biological fact that “most tissues have an *intrinsic* asymmetry, a polarity”. On the basis

of reversal experiments of D-V orientation, he thus contended that “such polarity proper would exist in many tissues in which it would occur as a very stable graded property which orients a generating activator maximum according to the internal polarity” (see 3 Figs in Meinhardt, 1982, in **I**).

2) From Slack's (1976, in **I**) graft experiments on the antero-posterior (A-P) organization of amphibian limbs, Meinhardt also admitted that there is strong evidence that “polarity does *not* result from many polar substructures but from the slope of graded distributions of morphogenetic substances”. Many of these so-called morphogens have now been detected and characterized (see retinoids, p. 443, 447).

Recent knowledge of the polar structures of the two ubiquitous self-assembling protein systems present as pools within the ground plasm, the tubulin and the actomyosin systems, contributes to bridge the gap between these two points of view, electric-structural (1) and gradiential diffusion of morphogens (2). The establishment of a cell polarity can thus be traced back to polarity of its molecular units, such as the tubulin heterodimer and the F-actin molecules interconnected by bipolar myosin-aggregates. Microtubules are the cytoskeletal elements actively determining biopolarizing processes through the active mechanical mediation of motor proteins such as dynein and kinesin (see IV.E.4) while actin microfilaments function as electric cables through ionic tunneling processes (see **I**, 1989) and confer polarity through their directional assembly processes controlled by positional DNA.

Positional information has been suggested to play a central role in developmental pattern formation and has recently been revisited by its proponent, Wolpert (1989), who distinguishes a positional signal from an inductive interaction because “the former specifies multiple states, confers polarity, and can act over a long range; a gradient in a diffusible morphogen is just one way of specifying position”. As directional polarities of cytoskeletal microfibrillar proteins must also be coded by positional sequences of the double-stranded DNA as suspected in the process of bipolar axiation of our *Allomyces* model (see **I**), we can then suggest that the DNA controlled polar orientation of morphogenetic gradients is primarily mediated by the polar positioning of protein filamentous structures endowed with *dual*, electric-structural polarizability.

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